RACE AND RACIAL DIFFERENCES

A Handbook for the 21st Century

How DNA Shapes Mankind into Seven Major Races

With 64 photographs, 16 illustrations, 7 maps, 6 tables, and 11 charts.

Arthur Kemp B.A.

Ostara Publications

Race and Racial Differences
A Handbook for the 21st Century
How DNA Shapes Mankind into Seven Major Races

By
Arthur Kemp B.A.

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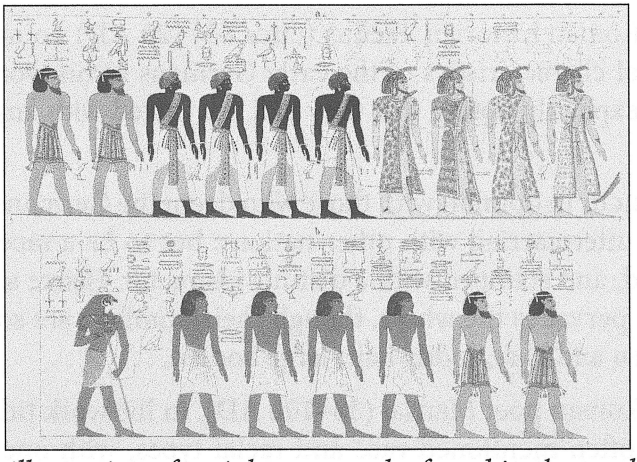
CHAPTER ONE
AN INTRODUCTION TO RACE

The division of humans into groups based on their physical appearance is as old as humanity itself. From the earliest societies onward, people have been able to distinguish "outsiders" by their physical appearance alone. For this reason, it has always been the standard way to differentiate between nations, and, indeed, up to a century or so ago, anyone claiming that race did not exist would have literally been thought of as delusional.

Racial Descriptions from Ancient Egypt

The first written references to race as a physical division of humankind date from Ancient Egypt. Numerous surviving depictions and stelae — including orders regulating the passage of black Nubians up the Nile — can be found in abundance.



An illustration of racial types can be found in the tomb of Pharaoh Seti I, Room D, at Thebes, Egypt (circa 1290 BC).

However, perhaps one of the most forthright descriptions of race can be found in the 19th Dynasty (1292 BC to 1189 BC) buildings at Thebes, which provides illustrated examples of how the Egyptians classified races.

Racial Descriptions from Classical Greece and Rome

Classical Greek writers also provided many references to racial differences. The physician Adamantius (circa 415 AD) wrote that the Greeks prided themselves upon possessing the "fairest eyes of all the nations" in his work *Physiognomica*, iii. 32.

The Greek philosopher Xenophanes (circa 570–478 BC) wrote that the "Ethiopians make their gods black and snub-nosed; the Thracians say theirs have blue eyes and red hair" (*Fragments*, B16. "Clement of Alexandria").

One of Classical Greece's most famous philosophers, Aristotle (384–322 BC), in his work *On the Generation of Animals*, remarked that "Further, children are like their more remote ancestors from whom nothing has come, for the resemblances recur at an interval of many generations, as in the case of the woman in Elis who had intercourse with the Aethiop; her daughter was not an Aethiop but the son of that daughter was" (*On the Generation of Animals*, 1.18.722A).

The Roman historian Tacitus (circa 65–120 AD) pointed out the racial characteristics of the early Germans in his work *Germania*, explicitly noting that they had not mixed with any other races:

"I concur in opinion with those who deem the Germans never to have intermarried with other nations; but to be a race, pure, unmixed, and stamped with a distinct character. Hence a family likeness pervades the whole, though their numbers are so great: eyes stern and blue; ruddy hair; large bodies . . ."

The Roman poet Martial (38–104 AD), in his work titled *Epigrams*, often mentioned race and racial types, with one of his most pointed remarks in Book VI, 39, where he tells of a Roman

woman who had had children by numerous men of different origins: "One of them, with woolly hair, like a Moor, seems to be the son of Santra, the cook. The second, with a flat nose and thick lips, is the image of Pannicus, the wrestler . . . of the two daughters, one is black . . . and belongs to Crotus, the flute player."

Confusion between Race and Nationality

The period following the end of the Roman Empire up to the Industrial Revolution continued to see many famous writers remark upon the fact that different races inhabited the earth, but, as time went on, the word "race" became used to describe what were, in fact, ethnic or linguistic groups, rather than groups of physically similar people.

In this way, many spoke of the "British race" or the "French race" — as if there was a uniform look and attitude inherent in the populations of those countries.

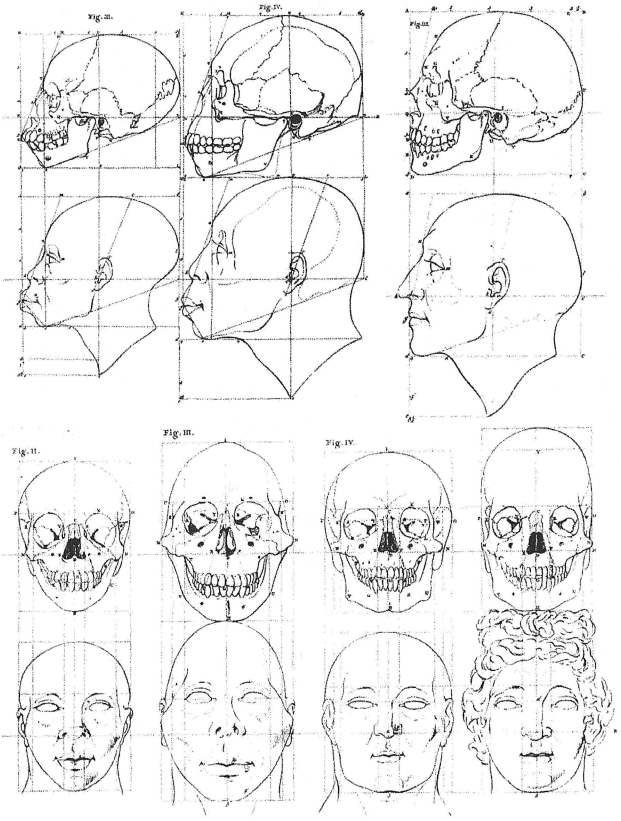
This misconception carried on for centuries, and it was only with the advent of the first scientific approaches to biology that a move away from this ignorant assumption was made.

Physical Appearance Race Classification Starts in 17th Century

The French physician François Bernier (1625–1688) provided the first scientifically-based racial classification.

Drawing upon the limited geographical knowledge of his time, he used the "four quarters" of the globe as his basis of classification, breaking humans down into "Europeans," "Far Easterners," "Negroes," and "Lapps" (*Nouvelle division de la terre par les différentes espèces ou races l'habitant,* "New division of Earth by the different species or races which inhabit it," 1684).

The next innovator in the study of racial differences was the Dutch surgeon and anatomist Petrus Camper (1722–1789). He provided the first scientific analysis of skeletal racial disparities, and in particular, what he called the "facial line" — a clear and marked physical difference between the races.



Petrus Camper's "Facial Lines" marked a turning point in the scientific analysis of racial differences.

Next was the famous Swedish botanist Carolus Linnaeus (1707–1778). He was the first scientist to formally classify all animal forms, a process he started in 1735.

His classification system — using Latin names — still forms the basis of modern biological naming systems. He included humans in his categories and introduced the term still used today: *homo sapiens.*

Linnaeus had four major human classifications: Homo americanus, Homo africanus, Homo europaeus, and Homo asiaticus.

The next scientist to attempt a categorization of the races was the German physician Johann Friedrich Blumenbach (1752–

1840). He produced a comprehensive overview of physical differences, based initially on a study of skull shapes.

His work, *Decas collectionis suae craniorum diversarum gentium illustrate* ("The Illustrated Collection of Dozens of Skulls of Different Races"), was first published in 1790. It divided the human species into five races, which he called "Caucasian" (for the "white race"), the "Mongolian" (or the "yellow race"), the "Malayan" (or the "brown race" by which he meant Southeast Asian and Pacific Islanders), the "Ethiopian" (or the "black race," which included sub-Saharan Africans), and the "American" (or the "red race," by which he meant all Amerinds).

A selection of skulls from Blumenbach's work.

It was from Blumenbach that the term "Caucasian" has derived. He chose this name to represent his "white race" category because he believed that a skull from the Caucasus Mountains was the most anatomically perfect one in his collection. The word "Caucasian" has come to mean white or European, particularly in the present-day United States.

Several other scientists then went on to emulate the practice of dividing groups of people by physical appearance. Sometimes they would also ascribe psychological attributes to the races, although these all remained opinions of those who had had dealings with the "races" in question rather than through any serious analysis.

It was only with the publication of the work *Essai sur l'inégalité des races humaines* ("Essay on the Inequality of the Races") by the French nobleman Joseph Arthur, Comte de Gobineau (1816–1882), that the concept of psychological differences — that is, in temperament and intelligence — between the races was first em-

pirically contemplated. This was published in five separate parts from 1853 to 1855.

Gobineau's racial categories — "the black, the yellow, and the white" (*Essai sur l'inégalité des races humaines*, volume 1, chapter 16) — were further divided into sub-categories. These broad categories were however reflective of the geographical knowledge of his time and were not all inclusive. Gobineau's most lasting contribution to racial science was his elevation of the Nordic European subtype as the leading light in all civilizations. He was the first to popularize the term "Aryan" as a word for that group.

His fellow countryman, anthropologist Count Georges Vacher de Lapouge (1854–1936) enthusiastically supported Gobineau's Nordicism. His 1889 book *L'Aryen: son Rôle Social* ("The Aryan: His Social Role") mapped out what he claimed was the superiority of the dolichocephalic (that is, "long-headed") Aryan races to the brachycephalic ("round-headed") races.

Francis Galton Develops the Science of Eugenics

The Englishman Francis Galton (1822–1911) was a polymath: a statistician, sociologist, psychologist, anthropologist, explorer, geographer, and inventor — and a half-cousin of Charles Darwin.

Greatly influenced by his famous family member's book on evolution, Galton went on to provide the first concrete examples of the overriding factor of inheritance in determining ability and intelligence — and not just physical characteristics. His 1869 book, *Hereditary Genius,* remains the classic study in this field. He also coined the phrase "nature over nurture."

Galton also founded the science of eugenics, a now-often abused term that merely means the selective breeding of animal sorts to bring out their best qualities.

Eugenics is standard practice in the animal breeding profession to this day. Galton and others wanted this science applied to humans for their betterment.

First Accurate Scientific Racial Typology Works Emerge in 19th Century

Around this time, several leading academics from various countries produced extensive works on racial typology and classification. The most prominent of these works are:

- *Völkerkunde* (1874), translated into English as *The Races of Man and Their Geographical Distribution* (1876), by German anthropologist Oscar Peschel (1826–1875);
- *The Human Species* (1879), by French biologist Jean Armand de Quatrefages (1810–1892);
- *The Races of Man: An Outline of Anthropology and Ethnography* (1900), by French anthropologist Joseph Deniker (1852–1918);
- *The World's Peoples: A Popular Account of Their Bodily and Mental Characters, Beliefs, Traditions, Political and Social Institutions* (1908) by Irish anthropologist A. H. Keane (1833–1912); and
- *The Races of Man and Their Distribution* (1909) by British anthropologist Alfred Cort Haddon (1855–1940).

The American MIT graduate and Harvard University professor William Zebina Ripley (1867–1941) won fame for his far-reaching study of racial anthropology *The Races of Europe: A Sociological Study* (1899). This work, which was developed from several lectures he gave in the preceding years, proved to be one of the first major authoritative studies of racial differences.

His fellow American, Madison Grant (1865–1937), a Yale and Columbia University graduate, won fame for his environmental conservation efforts (he is credited with the saving of many species of animals, including the bison; the founding of the Bronx Zoo, the Save the Redwoods League, and Denali National Park in Alaska; and for creating the discipline of wildlife management).

However, it was for his racial studies that Grant is best remembered. His 1916 book, *The Passing of the Great Race*, dealt

forthrightly with racial differences and the coming extinction of the white Americans who had founded that nation.

His contemporary and fellow American Theodore Lothrop Stoddard (1883–1950), a Ph.D. graduate from Harvard, produced many works dealing with race, the most famous of which was titled *The Rising Tide of Color Against White World-Supremacy* (1920). With Grant, Stoddard's works significantly influenced the American government to introduce the 1924 immigration act, which favored new settlers from northwestern Europe. This law was only revoked in 1965.

In 1921, three German scientists, Dr. Erwin Baur (1875–1933), Dr. Eugene Fischer (1874–1967), and Dr. Fritz Lenz (1887–1976), together published a significant work on the biological basis of human inheritance titled *Menschliche Erblichkeitslehre* (translated into English as *Human Heredity* and published in 1931). This extensive work also defined six major races, with subdivisions, along with a detailed explanation of how human traits were inherited.

German professor Hans Friedrich Karl Günther (1891–1968) produced his first work, *Rassenkunde des deutschen Volkes* ("Racial Science of the German People") in 1923. He went on to write overviews of other races as well, with his 1929 classic *Rassenkunde Europas* being translated into English *as The Racial Elements of European History.*

In that work, he defined race as follows: "A race shows itself in a human group which is marked off from every other human group through its own proper combination of bodily and mental characteristics, and in turn produces only its like."

Back in America, Carleton Stevens Coon (1904–1981), a Harvard University lecturer and president of the American Association of Physical Anthropologists, was tasked with producing an update to Ripley's classic work. Coon's 1939 edition of *The Races of Europe* contained not only updates to racial classifications but also a section that defined Jews as a distinct race for the first time.

Coon's 1962 work, *The Origin of Races*, proposed that the races were of different genetic origins (also called "multiregional evolution") and not of a single source, as others had claimed.

By the early 1930s, the ideas of race and eugenics had become commonplace in the academic world. An international eugenics society came into existence and held conferences in New York City, attracting delegates from all over the world.

Race-Denial Emerges after 1945

The adoption of racially based policies by Nazi Germany in 1933 also greatly boosted the study of race across the world. This turned out to be short-lived, as the defeat of Nazi Germany in 1945 and the subsequent relentless propaganda against that state led to an almost complete collapse in the study of race and racial differences.

This, in turn, opened the way for "race-denial," an ideology founded by the Jewish anthropologist Franz Boas (1858–1942), to take center stage in the academic world. This race-denial belief initially argued that racial differences were not inherent (that is, inherited). Today this belief has moved on and now denies that race even exists, except as a "social construct."

Nevertheless, a few writers continued to publish studies on race and racial differences. Perhaps one of the most prominent of these was Carleton Putnam (1901–1998), a Princeton and Columbia graduate.

An astute businessman, he founded an airline which eventually became known as Delta Air Lines. He served as chief executive officer of that company and held a seat on its board of directors until his death.

It was, however, the rise of the "Civil Rights" era in America which saw Putnam galvanized into writing *Race and Reason: A Yankee View* (1961), which was made required reading for high school students in Louisiana. He followed this up with his second book, *Race and Reality: A Search for Solutions* (1967).

Science of IQ Testing Enters the Mainstream

Audrey Mary Shuey (1900–1977) was Chair of the Department of Psychology at Randolph-Macon Women's College. In 1958, she published the first edition of her magnum opus, *The Testing of Negro Intelligence.* This work, which summarized the results of 40 years of intelligence tests in America — including from official US Army sources — revealed that there was at least a 15-point Intelligence Quotient (IQ) gap between blacks and whites, even when all environmental factors were accounted for.

In Britain, Oxford Emeritus Reader Professor John Baker (1900–1984) published his greatest work, titled *Race,* in 1974. That book, the last genuinely exhaustive racial typology work ever published by the "mainstream" publishing houses, contained an overview of all the races of the earth and ended with a review of race and achievement, which concluded that no truly indigenous civilization had ever arisen in sub-Saharan Africa.

Political pressure from the race-denial lobby had by now started to intensify, and Oxford University Press, which had published Baker's book, was forced to withdraw it from circulation. It was then republished in America by a group of free speech activists calling themselves the Foundation for Human Understanding (FHU). From there, it went out of print once again.[1]

Racial typology was thereafter completely suppressed as a science by the race-denying "mainstream" publishing world and demonized in the mass media.

Race and IQ were still discussed, mainly because the evidence was so overwhelming that even race deniers could not pretend it did not exist. Instead of denying its existence, the race denial lobby sought to explain the persistence of the IQ gap between the races as purely the result of environment.

The simmering debate came to a dramatic head with the 1994 publication of *The Bell Curve: Intelligence and Class Structure in*

1 Baker's book has since been republished by Ostara Publications, which acquired the rights from FHU.

American Life, written by psychologist Richard J. Herrnstein and political scientist Charles Murray.

In this book, Herrnstein (whose Jewishness served to deflect the by now standard accusation of "Nazi" used against any work discussing race) and Murray showed that human intelligence is primarily inherited. The work also showed that IQ levels are an extremely accurate predictor of behavior, including financial status, social responsibility, out of wedlock birth and criminality, among a host of factors. The book was a commercial success despite the still-ongoing efforts by race-denialists to denigrate it, and the "bell curve" has entered the popular idiom as an expression of IQ.

Further work on the topic of IQ and race was, however then formally driven from the "mainstream" publishing houses. Other books on racial typology, race, and IQ have since then only been published privately, and censored off the dominant commercial bookselling platforms.

The only recent exception to this rule has been a book by the British-born former science and health editor for *The New York Times*, Nicholas Wade. His work, titled *A Troublesome Inheritance: Genes, Race and Human History* (2014), dared to point out that genetics had significant implications for the social sciences. This relatively mildly phrased book provoked a hysterical reaction from the race-denial lobby, and included a public rejection published in the *New York Times Book Review.* As was now usual, the criticism did not address any of the facts quoted in the book — many from the same people who later condemned the work — but instead focused on ad hominem attacks upon the author personally.

The race-denialist faction seemed supreme: they had conquered the academic world. They expelled any sociologists who disagreed with the Boas worldview that race did not exist and had almost succeeded in driving any further race-related research underground.

The emergence of race denial as the dominant theory of the late 20th Century and the early 21st Century was supposedly cemented by official declarations from the major academic bodies — including the American Anthropological Association — that race was a "social construct" and did not exist as a biological reality.

Emergence of Genetic Science Undercuts Race-Denial Theory

However, the emergence of the science of genetics has proven to be an unwelcome thorn in the side of the race denialists.

The history of the study of genetics started in 1866, when a German monk by the name of Gregor Mendel (1822–1884) conducted experiments with pea plants in his care. He worked out, through careful observation, the basic rules of heredity. This discovery, which largely went unnoticed at the time, is now referred to as the laws of Mendelian inheritance.

In 1869, a Swiss chemist and biologist named Friedrich Miescher (1844–1895) was studying white blood cells in his laboratory when he identified phosphate-rich chemicals in the blood cell's nuclei. He called these chemicals "nuclein." They are now known as deoxyribonucleic acid, or DNA.

Miescher was aware of the significance of his discovery and wrote at the time that these chemical compounds were linked to the process of heredity.

In 1900, Mendel's work was rediscovered and republished independently by several scientists in Austria, Germany, and the Netherlands. By 1902, the English physician Archibald Garrod (1857–1936) had shown how Mendelian inheritance laws were applicable to humans through his discovery of the inheritance of disease.

Garrod's book, *The Incidence of Alkaptonuria: A Study in Chemical Individuality* (1902), was the first work to show that alkaptonuria was an inherited recessive disorder whose outcome could be predicted in humans by studying their lines of inheri-

tance. The impact of this finding — nowadays regarded as commonplace — was sensational at the time, as it opened the door to understanding inheritance laws in humans.

In the years following, scientists turned their attention — and their ever more powerful microscopes and analytical equipment — to studying human cells and their inheritance characteristics.

DNA Isolated for the First Time in 1944

Finally, in 1944, a Canadian American immunochemist at the Hospital of the Rockefeller Institute for Medical Research, Oswald Avery (1877–1955), isolated DNA as the material of which genes and chromosomes are made.

He made this breakthrough while studying the bacterium responsible for pneumonia, called pneumococcus. One of his experiments revealed that if a live form of pneumococcus was mixed with an inert but lethal form of that bacteria, the previously harmless form would soon become deadly.

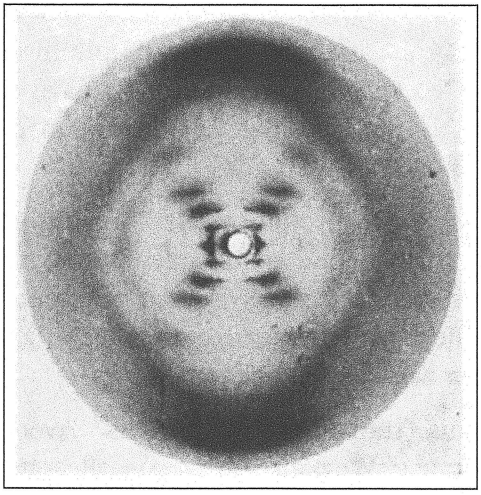
Baffled by this "transformative process," Avery determined to find out its cause. Working with two colleagues, Colin MacLeod and Maclyn McCarty, he eventually determined that the "transforming substance" was a nucleic acid or DNA. The three scientists published their findings in the *Journal of Experimental Medicine* in 1944.

When a professor of biochemistry at Columbia University medical school, Erwin Chargaff (1905–2002) — an Austrian Jew who had emigrated to America before World War II — read Avery's paper, he decided to start research on the chemistry of nucleic acids.

Finally, by 1950, after much detailed work, Chargaff was able to reveal two significant findings: (1) that in any double-stranded DNA, the number of chemical units on either side of the strand was always equal, and (2) that the composition of DNA varies between species. These discoveries are now known as "Chargaff's Rules."

DNA Molecule Photographed and Described 1952–1953

The next important breakthrough was made by British scientist Raymond Gosling (1926–2015). While working as a Ph.D. student at King's College, London, he took several X-ray crystallographic images of DNA. The clearness of these images provided the first visual look at DNA, and one of his photographs (known as "photo 51") became world-famous.



Raymond Gosling's famous Photo 51, showing X-ray diffraction pattern of DNA.

Gosling's academic supervisor, Rosalind Franklin (1920–1958), had helped him perfect the X-ray crystallographic process. She now also worked with him to calculate the dimensions of the DNA strands and venture the opinion that phosphates were on the outside of what was probably a helical structure.

These conclusions and photographs were used by three scientists to first outline the double helix structure of the DNA molecule. These three scientists — the British molecular biologist Francis Crick (1916–2004), the American molecular biologist James Watson (born 1928, age 94 in 2022), and the New Zealand-born British biophysicist Maurice Wilkins (1916–2004), all received the 1962 Nobel Prize in Physiology or Medicine for this discovery.

Watson later served as director of Cold Spring Harbor Laboratory (CSHL), and under his direction, it became one of the world's leading research centers for molecular biology.[2]

Genetic Code Discovered in 1961

In 1961, German biochemist Johannes Heinrich Matthaei (1929, age 94 in 2022), while working as a post-doctoral student in the Bethesda, Maryland, National Institutes of Health (NIH) laboratory, discovered the set of rules (known as the "genetic code") used by living cells to translate information encoded within genetic material into proteins.

Controversially, the head of the laboratory where Matthaei made the breakthrough, an American-Jewish biochemist by the name of Marshall Nirenberg (1927–2010), and two other research associates, India-born Har Gobind Khorana (1922–2011) and Illinois-born Robert Holley (1922–1993), were awarded the 1968 Nobel Prize in Physiology or Medicine for this discovery.

The three prize-winners — Nirenberg, Khorana, and Holley — all had made significant contributions to the solution of the genetic code in their own right, but the denial of the Nobel Prize to Matthaei remains unexplained to the present day.

DNA Sequencing Discovered 1977

In 1977, another turning point was reached when British biochemist Frederick Sanger (1918–2013) discovered a method whereby the order in which DNA's four chemical building blocks, called "bases," could be calculated. This is known as "DNA sequencing," and the process whereby this happens is called the

2 Between 1988 and 1992, Watson was associated with the National Institutes of Health, helping to establish the Human Genome Project, which completed the task of mapping the human genome in 2003. After serving as president and chancellor of CSHL, he was finally forced to resign in 2007 after pointing out in public that there was a genetic link between intelligence and race. When he repeated his views in a television documentary in 2019, CSHL revoked his honorary titles and severed all ties with him — a stark indication of how powerful the race-denialist lobby had become, and of how fearful they were of the implications of genetic research.

"Sanger method." This breakthrough, for which Sanger received a Nobel prize, allowed the entire human genome to be sequenced.

Polymerase Chain Reaction (PCR) Analysis Invented 1983

In 1983, a method of analyzing the composition of DNA through a small sample (such as a cheek swab or a small amount of saliva) rather than laborious blood sampling, was developed. This process, called Polymerase Chain Reaction (PCR) was invented by American biochemist Kary Mullis (1944–2019) and British biochemist Michael Smith (1932–2000). They were jointly awarded the Nobel Prize in Chemistry in 1993 for this breakthrough.

Human Genome Project: Launched in 1988, Completed in 2013

In 1988, the Human Genome Project was officially launched. This US-government sponsored project was tasked with determining all 3.2 billion chemical components, or "letters," of human DNA.

By 1999, the entire genetic code of one human chromosome, number 22, was unveiled. It contained 33.5 million "letters." The Human Genome Project was finally completed in 2003.

Single Nucleotide Polymorphisms (SNPs) Analysis Developed

The next decade saw even further advances, with geneticists able to study tiny areas within the DNA structure called Single Nucleotide Polymorphisms (SNPs). This proved to be a significant breakthrough, as SNPs were shown to play a major role in determining ancestry, and predispositions to genetic illnesses.

Commercially Available DNA Ancestry Home Testing Kits Emerge

Sequencing processes continue to improve, and this technology lies at the heart of present-day "DNA testing kit" companies, which specialize in ancestry prediction and health proclivities based on an individual's submitted DNA sample.

Ancestry testing is becoming increasingly refined with each passing year. It is now, in many cases, able to pinpoint specific counties or regions of origin within countries.

DNA Testing Companies Divide Globe into Seven "Major Populations"

For example, one of the widely known ancestry testing companies, 23andMe,[3] lists the following seven major "global populations" as:

"European, Central & South Asian, Indigenous American, East Asian, Sub-Saharan African, Western Asian & North African, [and] Melanesian."[4]

In other words, simple "at home" DNA tests allow for the categorization of individuals, based on a saliva sample, into at least one of seven major identifiable racial groups.

As if this was not enough of a problem for the race-denialists, detailed SNP testing also allows for a regional breakdown of these results. This allows individuals to find out specific regions from where their ancestors came.

To continue with the *23andMe* example, that company offers SNP analysis, which can further predict specific regions within these seven major divisions:

European Group:

Northwestern European: British & Irish, Finnish, French & German, Scandinavian, [and] Broadly Northwestern European.

Eastern European: Poland, Belarus, Czech Republic, Estonia, Hungary, Latvia, Lithuania, Russia, Slovakia, Slovenia, [and] Ukraine.

3 Important: The mention of any commercial DNA testing company in this book does NOT imply or contain any link or endorsement of any sort between that company and this book, its author, or publisher.

4 *23andMe Customer Care, Reports, Ancestry, Composition*, "23andMe Reference Populations & Regions," retrieved from the 23andMe website, September 2022.

Southern European: Greek & Balkan, Italian, Sardinian, Spanish & Portuguese, [and] Broadly Southern European.

Central and South Asian Group:

Central Asian, Northern Indian, & Pakistani: Bengali and Northeast Indian, Central Asian, Gujarati Patidar, [and] Northern Indian and Pakistani.

Southern Indian Subgroup.

Southern South Asian, Malayali Subgroup, [and] Southern Indian & Sri Lankan.

Indigenous American Group:

Alaska, Columbia River Basin, Great Basin & Lower Colorado Basin, Great Lakes and Canada, Northeast, Plains, South Central, [and] Southwest.

East Asian Group:

Chinese: Northern Chinese & Tibetan; Southern Chinese & Taiwanese; South Chinese; Vietnamese; Filipino & Austronesian; Indonesian, Thai, Kymer, & Myanma; Chinese Dai; Japanese, [and] Korean.

Northern Asian: Manchurian & Mongolian, Siberian, [and] Broadly Northern Asian.

Broadly East Asian.

Sub-Saharan African Group:

West African: Ghanaian, Liberian & Sierra Leonean, Nigerian, [and] Broadly West African.

Northern East African: Sudanese, Ethiopian & Eritrean, Somali, [and] Broadly Northern East African.

Congolese & Southern East African: Angolan & Congolese, Southern East African, [and] Broadly Congolese & Southern East African.

African Hunter-Gatherer: Pygmy and San peoples of central and southern Africa.

Western Asian & North African Group:

Arab, Egyptian & Levantine: Coptic Egyptian, Egyptian, Levantine, Peninsular Arab, Broadly Arab, [and] Egyptian & Levantine.

North African: Algeria, Libya, Morocco, [and] Tunisia.

Northern West Asian: Anatolian; Cypriot; Iranian, Caucasian, & Mesopotamian, [and] Broadly Northern West Asian.

Melanesian Group:

Fiji, [and] Denisovan DNA.

In addition, 23andMe has an "Ashkenazi Jewish" category, which they differentiate from the "Levantine" category by virtue of the presence of some European markers which represent a bottleneck admixture of certain Europeans and Jews in Eastern Europe.[5]

Home DNA Ancestry Testing Based on Biological Basis of Race

23andMe is of course not the only commercial DNA ancestry testing service. There are a great many such services now in operation, and all follow more or less the same categories. The fact that such categorizations can be made using saliva sample DNA tests is directly contradictory to the race-denialists' claim that there is no biological basis to racial categories.

In fact, the ability to determine racial origins by DNA samples is absolute, conclusive, and irrefutable proof that there is a biological basis to race, and that these traits are obviously inherited.

Physical Features Contained within DNA Samples, Forensics Reveals

It is not only possible to determine racial and regional origin from tiny DNA samples, but detailed analysis of SNPs allows forensic scientists to recreate facial features, skin types, hair color, hair type, and all other aspects of a selected individual.

5 All these categories are from 23andMe fact sheets.

This sort of DNA analysis is used by police departments around the world in order to identify suspects, solve crimes,[6] and even produce "identikits" of decomposed bodies.[7]

There are a number of commercial companies offering such services. One example is based in the US state of Delaware, and trades under the name Parabon Nanolabs.[8]

This company specializes in assisting law enforcement in solving crimes by using the fact that SNP analysis can accurately predict the appearance of an individual based on genetic markers alone.

Law Enforcement uses DNA to Identify Race and Create Identikits

Some examples of the ability to identify race and physical appearance and racial origin from small DNA samples alone are apparent from the illustrations on the next two pages, as used by police departments throughout the world, and by Parabon Nanolabs to promote their services.

Parabon Nanolabs bases its classification process on the division of humans into what it calls "seven continental populations: African, Middle Eastern, European, Central/South Asian, East Asian, Oceanian, and Native American" and they state that this is possible because of the detailed analysis of SNPs on an individual sample.[9]

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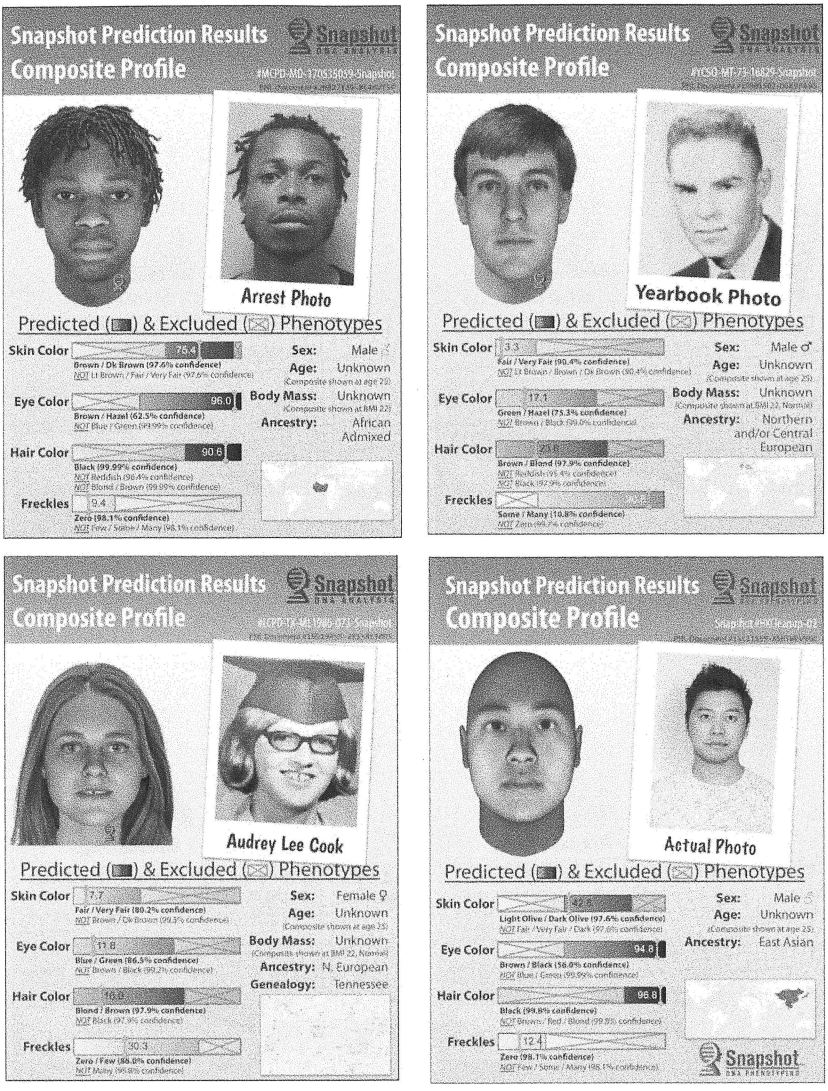
6 "How new forensic science is helping police solve cold cases like the Golden State Killer," *New York Post,* September 9, 2022.

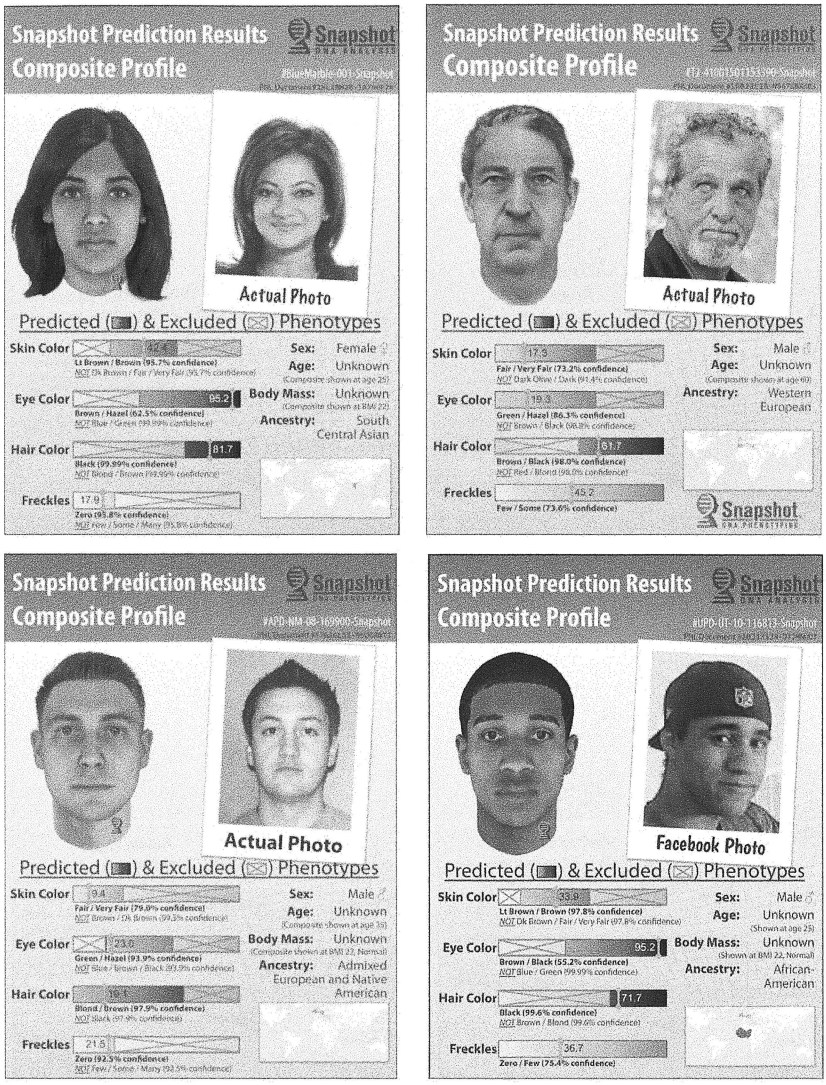
7 "DNA helps Florida investigators ID victim 41 years later," *The Associated Press*, July 22, 2022.

8 Once again, it is important to state that the mention of Parabon Nanolabs as an example of the sort of company which provides law enforcement with DNA phenotyping services in this book is *purely random.* There are other companies and law enforcement agencies which provide the same services. It is important *to note there is no connection, endorsement, or any other sort of link between Parabon Nanolabs and this book, its author, its publisher, or any other entity.* All material used here is in the public domain.

9 "Predicting Genetic Ancestry With Snapshot," Parabon Nanolabs, retrieved September 2022 from snapshot.parabon-nanolabs.com/ancestry.



Above: The race and physical appearance of (from left to right, top to bottom) an African (admixed), a Northern/Central European, a Northern European, and an East Asian (Chinese) were all accurately predicted from small DNA samples alone. Race and physical appearance can be determined by analyzing Single Nucleotide Polymorphisms ("SNPs") from tiny amounts of genetic material. The predicted race and physical appearance is on the left of each image, and the actual subject is on the right.



Above: The race and physical appearance of (from left to right, top to bottom) an Indian (South Central Asian); a Western European; a European admixed with Amerindian; and an African (admixed) were all accurately predicted from small DNA samples alone. Race and physical appearance can be determined by analyzing Single Nucleotide Polymorphisms ("SNPs") from tiny amounts of genetic material. The predicted race and physical appearance is on the left of each image, and the actual subject is on the right.

Genetics Makes a Final Definition of Race Possible: A Group with Common DNA

There can therefore no longer be any question that race, and racial differences, are genuine biological realities which are observable to the naked eye, and which are genetic in origin.

Exactly how genetics and DNA form races, and a description of these major racial groups of the world, makes up the rest of this book.

Here, however, it can be definitively said that the definition of race is as follows: **a closely related group of people whose common physical appearance (and, as will be shown, psychology) are determined by genetic factors alone.**

CHAPTER TWO
THE STRUCTURE OF HUMAN DNA

Anyone seeking to understand how genetics creates races must first have a proper understanding of the structuring of human genetics. For this purpose, it is necessary to ensure that the reader has a clear grasp of all the major terms which will be encountered in any discussion of genetics, and specifically how races are created. The following overview is therefore critical to any such understanding, and the reader is urged not to skip this chapter, as it lays the framework for understanding the permanence of racial characteristics, which are the product of inheritance alone.

The Human Cell

The cell is the basic building block of all live organisms, and the human body is no exception. The human cell is tiny — about 100 micrometers in size (that is one thousandth of a millimeter, or about 0.00004 inch). There are around 30 trillion cells in an average human body.

There are a number of parts to a cell, but for an understanding of genetics, the nucleus is the most important. The nucleus is the part which contains the cell's hereditary material. This genetic material is the sole determinant of the nature and form of that cell's inward and outward appearance, as all other functions of the cell — and the body — are coded by instructions given from this hereditary material.

Deoxyribonucleic Acid (DNA)

It is inside the cell that deoxyribonucleic acid, or "DNA," is found.

Broken down, this "DNA" is made up of the following components:

Nucleobases

Nucleobases are nitrogenous (that is, containing nitrogen) compounds. There are five *nucleobases* relevant to the understanding of human genetics. These five *nucleobases* are:

— Adenine (represented by the symbol "A"). This compound consists of five (5) carbon atoms, five (5) hydrogen atoms, five (5) nitrogen atoms, and one (1) oxygen atom.

— Cytosine (represented by the symbol "C"). This compound consists of four (4) carbon atoms, five (5) hydrogen atoms, three (3) nitrogen atoms, and one (1) oxygen atom.

— Guanine (represented by the symbol "G"). This compound consists of five (5) carbon atoms, five (5) hydrogen atoms, five (5) nitrogen atoms, and one (1) oxygen atom. This compound differs from adenine in the way the atoms are joined together.

— Thymine (represented by the symbol "T"). This compound consists of five (5) carbon atoms, six (5) hydrogen atoms, two (5) nitrogen atoms, and two (2) oxygen atoms.

— Uracil (represented by the symbol "U"). This compound consists of four (4) carbon atoms, four (4) hydrogen atoms, two (2) nitrogen atoms, and two (2) oxygen atoms. Uracil is only found in what is called "RNA" (see below).

Nucleotides

Nucleotides are molecules made up of the combination of:

— one of the *nucleobases*: A, C, G, T, or U; and

— a sugar molecule with five carbon atoms.

There are two types of sugar molecules in human DNA: Deoxyribose and ribose. Deoxyribose has five (5) carbon atoms, ten (10) nitrogen atoms, and four (4) oxygen atoms. Ribose has five (5) carbon atoms, ten (10) nitrogen atoms, and five (5) oxygen atoms. When the sugar molecule contains deoxyribose, it is found

in "DNA" and when it contains ribose, it is found in "RNA" (see below); and

— a phosphate, made up of one (1) phosphorus atom and four (4) four oxygen atoms. When attached to a molecule containing carbon, it is called a phosphate group.

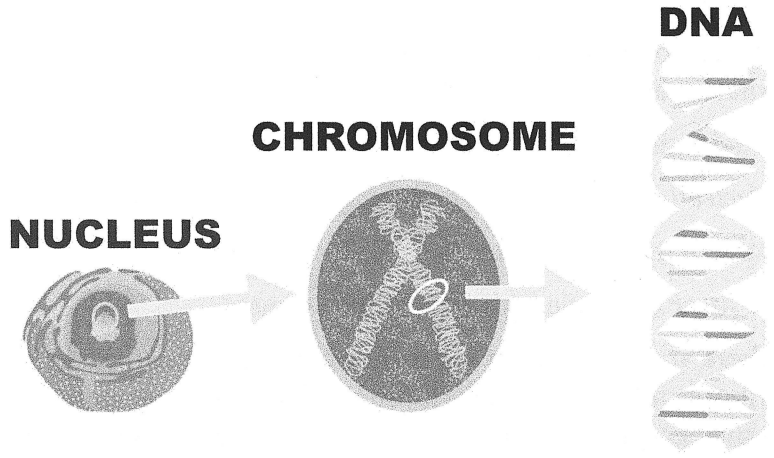
Polynucleotides

Polynucleotides are long chains of nucleotides, consisting of a "backbone" of sugar and phosphate molecules, with each of the sugar molecules linked to one of the nucleobases A, C, G, T, or U.

DNA is made up of long chains of polynucleotides.

Two polynucleotide strands that twist around each other to form a double-stranded helix. In the popular mind and the media, this helix formation is the most common representation of "DNA" and "genetics."

In this double helix — which can be described as having the shape of a "twisted ladder" — the nucleobases link across the two sugar and phosphate "backbones," or "strands," in a specific manner using hydrogen bonds. In this pairing process, the "C" nucleobase always pairs with the "G" nucleobase, and the "A" nucleobase always pairs with the "T" nucleobase.

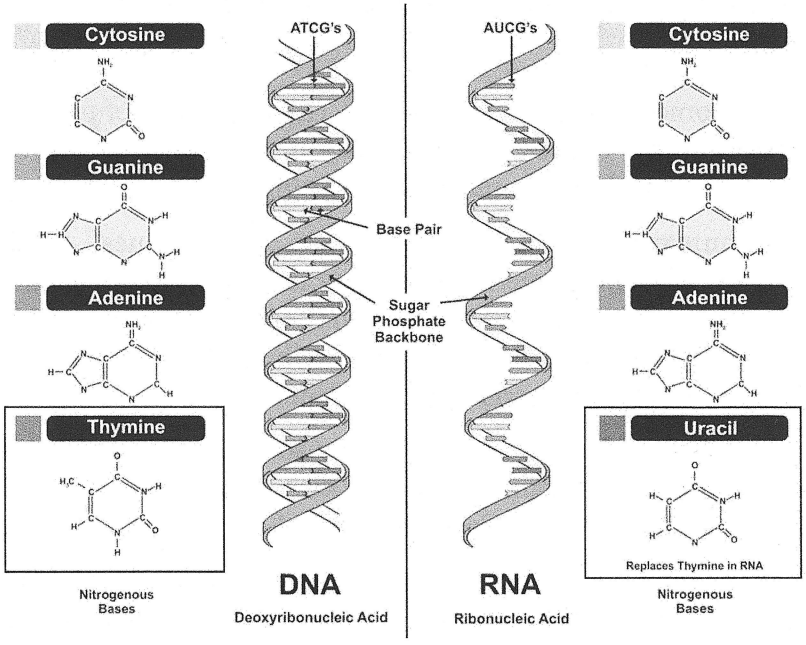


Nucleus to Chromosome to DNA

The "twisted ladder" structure allows it to be "copied" or "replicated" (by an enzyme known as DNA polymerase, which assembles the correct nucleotide bases, and builds duplicates using the existing "chain" as a template).

The "twisted ladder" also stores information in the linear sequence of the nucleotides along each strand. It is the order in which these "bases" (A, T, G, and C) are placed which constitutes the "genetic code" of a human.

This four-letter "alphabet" (A, T, G, and C) is arranged in specific formats, which are called "*codons*." These codons are the chemical composition of specific amino acids. For example, the codon "AGC" codes for the amino acid serine, and the codon "ACC" codes for the amino acid threonine.



A DNA strand (left), showing its four chemical components ("nucleobases") and how they are arranged inside the "double helix" shape. On the right, a RNA strand, with its four nucleobases attached to the single helix shape.

Ribonucleic acid (RNA)

One of the cell's components is called the "cytoplasm." This is an encased fluid which surrounds the nucleus. It contains Ribonucleic acid, or "RNA."

Just like DNA, this RNA is made up of a chain of nucleotides, except that it has the *nucleobase* Uracil ("U") instead of Thymine ("T"). RNA also has only a single strand structure, folded in on itself, and does *not* have the "double" or "ladder" structure of DNA.

RNA's purpose is to control the process through which proteins are generated (using a system called "messenger RNA" or mRNA, "transfer RNA" or tRNA, and Ribosomal RNA. RNA also controls the process known as "gene regulation," which controls when, how, and how much of a gene is "*expressed*" (activated).

Chromosomes

A chromosome is a strand of DNA (that is, chains of *polynucleotides* in the "twisted ladder formation") combined with proteins and packed tightly into self-containing units.

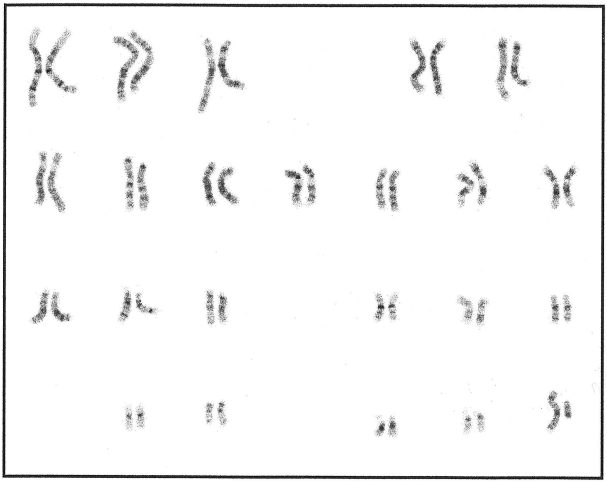
All humans have two (2) pairs of "sex chromosomes" — the DNA strands which determine whether that individual is a male or female, and two pairs of twenty-two (22) chromosomes which determine everything else about that individual. These non-sex determining chromosomes are called "autosomes."

The DNA molecules inside a chromosome are tightly wound around proteins (called *histone proteins*) which compress the DNA strands and help to determine how they behave.

There are five basic *histone proteins*, named H1 to H5. H1 and H5 are "linker histones" which act to link the DNA strands, and H2, H3, and H4 are the "core histones" around which the DNA is wrapped.

Each chromosome has a "p" arm and a "q" arm. These merely refer to the length of the segments, with the "p" (from the French word *petit*) being the shorter arm, and the "q" (so named only

because it is the next letter in the alphabet after "p") is the longer arm.



An image of a normal human male chromosome set, called a "karyotype"

Genes

A "gene" is made up of a series of *nucleotides* on a DNA strand at any specific place on a *chromosome.*

In other words, "genes" are areas on the chromosomes which are in control of certain functions. There are at least 20,500 genes in a human, and these genes vary in size between a few hundred DNA *bases* to more than two (2) million bases.

The actual location of a gene on a chromosome is called a "locus" (*loci* in plural).

Each individual has two copies of each gene, one inherited from each parent.

Genes consist of three types of *nucleotide* sequences:

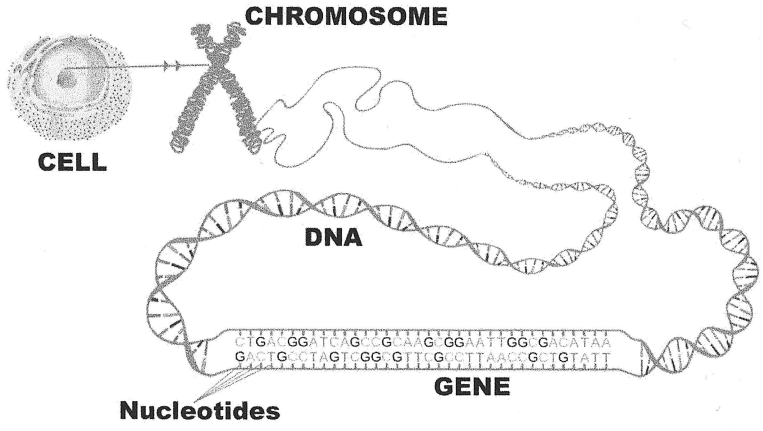
- coding regions, called "*exons*," which specify a sequence of amino acids.

- non-coding regions, called "*introns*," which do not specify amino acids, but play a vital role in the manufacture and the quantity

of the proteins in a gene. They also provide "space" for new exons to be added into the DNA sequence.

- regulatory sequences, which play a role in determining when and where the protein is made (and how much is made).

In addition, there are what are called "*pseudogenes*," which are segments of DNA that are "extra" copies of other genes, but which are characterized by a lack of sequences required to being them to expression.



Nucleus to Chromosome to DNA to Gene

Alleles

When any one of two or more genes occur at a given site (*locus*) on a chromosome, they are called alleles.

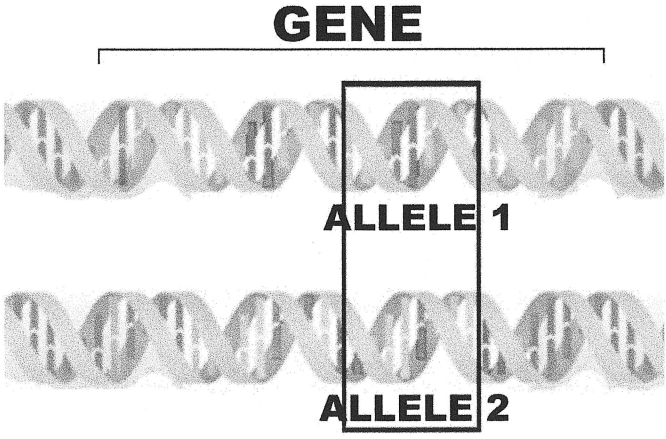
Alleles may occur in pairs, or there may be multiple alleles affecting the "expression" of a particular trait. This is called the "genotype," or the genetic constitution of an individual.

If the paired alleles are the same, the genotype is called "homozygous," and if they are different, it is called the genotype is called "heterozygous."

An allele is therefore a variant form of a gene.

An allele is called "dominant" when it overrides the traits of other alleles in a heterozygous pairing. When an allele is "recessive," it means that it will always be overridden by a dominant allele in any heterozygous pairing.

A given gene may have multiple different alleles, though only two alleles are present at the gene's locus in any individual.



The position of a gene, and an allele on a DNA strand.

Alleles are of vital importance in human genetics. They play a major role, along with SNPs (below) in determining all the traits of an individual, both physical and psychological.

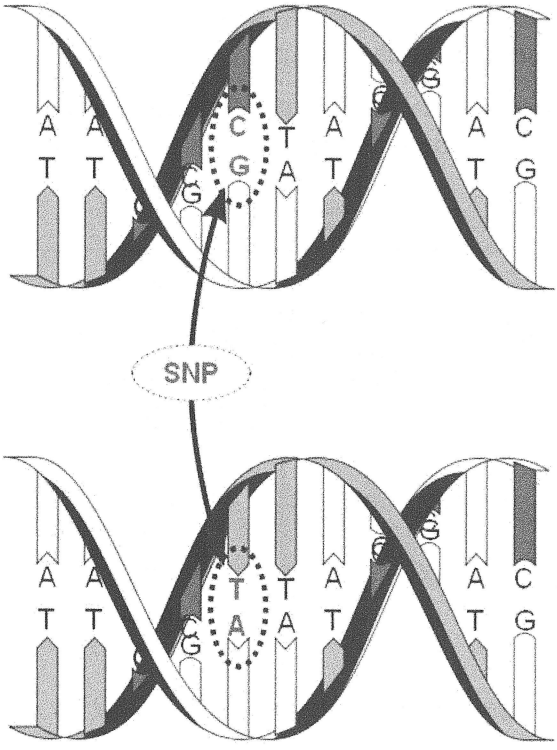
These trait-determining alleles may occur in pairs or be in multiple arrays. No matter how they are linked, it is these alleles which determine the *expression* of the gene: in other words, how it is shaped and created.

When this expression takes the form of the physical appearance of a trait, the result is known as a *phenotype*. This is the outward physical appearance of that individual.

Single-Nucleotide Polymorphisms (SNPs)

A "*single-nucleotide polymorphism*" (abbreviated to "SNP," and often spoken of as a "*snip*") is a variant at a single base position

in DNA structure, located at the point where two *nucleobases* (A and C, or G and T) link up.



The position of a single-nucleotide polymorphism (SNP) inside a DNA strand.

SNP variations are the most common sort of genetic variation between people, and affect all traits, physical (appearance and health), and psychological (character and cognitive ability).

Each and every SNP represents a difference in a single *nucleotide.* This can, for example, mean the replacement of the "C" *nucleotide* with the "T" *nucleotide* in a certain stretch of DNA. This change then alters the entire "expression" or output of that particular gene.

SNPs play a major role in ancestry determination, because individuals from closely related groups have similar SNPs.

This means that the more closely related individuals are, the more SNPs they will have in common.

Haplotypes: X, Y, and mtDNA

Each human being possesses some alleles which always remain the same and are never subjected to any sort of change. These alleles are called *haplotypes*.

Two of these allele groups — the "X" and the "Y" — form chromosomes by themselves, and are, like the other chromosomes, located inside the cell.

The X and Y chromosomes serve a primary function in determining the sex of the individual, with the X chromosome being the female chromosome, and the Y chromosome being the male counterpart.

The Y chromosome contains at least 55 genes, while the X chromosome contains at least 900 genes. A human egg cell, as carried in a female, always has an X chromosome.

Sex is determined at the moment of conception, depending upon the chromosome being carried by sperm cell. If the latter carries an X chromosome, the fetus (which already has a "built-in" X chromosome) ends up with two X chromosomes (XX) and will therefore be female. If the spermatozoon carries a Y chromosome, then the fetus will end up with an X and a Y chromosome (XY) and be a male.

This simple process carries with it an important genetic implication: namely that Y chromosomes are therefore passed down unchanged from father to son.

As a result, the study of Y chromosomes contains a valuable insight into the origin of that individual in terms of descent on the paternal side.

The X chromosome, however, will also include DNA from some of the female's paternal ancestry and is therefore less reliable in determining a solely maternal ancestral descent.

This is where another group of alleles, known as "mitochondrial DNA," or mtDNA, plays an important role. Mitochondrial DNA is found outside the nucleus of the cell, unlike the chromosomes mentioned above, which are located inside the cell nucleus.

mtDNA serves primarily as the "energy source" for the cells, but, because they are exclusively inherited through the female line, serve the same purpose in tracking ancestry as Y chromosomes do for the male. mtDNA is also categorized as a haplotype.

Haplogroups

When individuals are closely related to each other, they will all share a certain haplotype — most likely from a single, or small group of ancestors, with a unique SNP mutation.

Y haplogroups and mtDNA are reliable indicators of line of descent — and therefore of an individual's original founding group.

Autosomal DNA (atDNA)

Autosomal DNA is quite simply all the DNA which is not sex-determining in nature. This means that all DNA which is not part of the X or Y chromosomes are known as "autosomal DNA."

Autosomal DNA, containing as it does, the vast majority of genetic material, SNPs and alleles, provides a wealth of information about an individual, including origin, background, physical characteristics, and so on.

Individuals sharing a lot of autosomal DNA are also, obviously, closely related to one another. This fact also points toward "groups" of people, or "families" of people.

How Haplogroups Are Named

Just like X and Y chromosomes, the *autosomal* haplogroups (that is, the non-sex determining haplogroups) have also been given alphabet "names" in order to differentiate them from each other.

In this way, Y chromosomes are named from A to R, and mtDNA haplogroups are named from A to Z.

X chromosomes are not numbered or categorized in the same way, as they are subjected to random mutation due to the fact that they are not solely inherited from the female line.

As there is obviously some duplication in alphabet names, it is important that the haplogroup name always indicates if it is "Y" or "mtDNA."

Haplogroup "Subclades"

Withing any given haplogroup, there are often small variations ("mutations") at the SNP level. When one of these variations occurs, the haplotype is given its own category, or subgroup which falls under the main haplogroup heading.

Each subgroup is given the name of the SNP mutation which occurs at the furthest point away from the allele's center. This "furthest" SNP is called the "terminal SNP."

These subgroups are given the name "subclades" or "clades."

Subclades are identified by name. The first letter of a subclade's name will be the haplogroup "alphabet name" of its "parent" haplogroup, followed usually by a hyphen and the name of the terminal SNP.

A practical example of this would be the Y chromosome called Haplogroup R. This haplogroup is divided into a large number of subclades, each of which is found in different regions where members of that group have settled. Names of some of the subclades of Haplogroup R include R0a, R1, R1a, R1a1: R2, R3, R5, R5a, R5a1, R5a2, and so on.

Identifying the Location of Specific Genes and Alleles

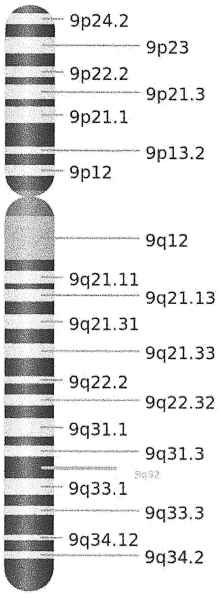
To aid scientists in the identification of specific genes, chromosomes have been allocated "addresses" based on:

– the chromosome's "number" (that is, chromosomes 1 to 22. The X and Y chromosomes do not have numbers.) and

– the sequence of nucleotides which make up the chromosome.

These nucleotide sequences are known as "bands," and are specific areas on the chromosome. These bands appear when the chromosome is stained in a certain way under analysis in a laboratory.

The first number or letter used to describe a gene's address represents the chromosome. The autosomal chromosomes 1 through 22 are designated by their chromosome number. The sex chromosomes are designated by X or Y.



The human chromosome 9, with some of the gene loci (the figures starting with 9p and 9q) highlighted.

Two examples will illustrate this system:

– The gene named "IGF2R," and specifically allele 5 (which is one of the important genes affecting intelligence) is located at the address "6q25-27."

This means that this gene is located on Chromosome 6, q-arm ("long" arm), bands 25 to 27.

– The gene named "CYP4B1" (which controls an individual's drug metabolism and synthesis of cholesterol, steroids, and other lipids) is located at 1p33.

This means that this gene is located on chromosome 1, "p" arm ("short" arm), band 33.

Factors Influencing "Genetic Diversity"

From the above, it should be clear to the reader that all these aspects of DNA are inherited, that is to say, passed down from parents to children. This always happens without change.

There are, however, some factors that can determine which traits are passed on, and which are not. The "loss" — or "addition" — of some traits leads to what is commonly called "genetic diversity" within a group.

There are five major factors which create genetic diversity: mutation, genetic drift, natural selection, migration, and non-random mating patterns.

- "Mutations" are the result of changes in the nucleotide sequence of a DNA strand. They come about due to errors in DNA while replicating, or through outside environmental influences such as chemicals.

These mutations can have a "knock on" effect on the manner in which the genes are "expressed," due to the changed codon. One example of this is sickle cell disease — found in those of African descent. This is what is called a "point mutation." This mutation is passed on through inheritance, like all other traits.

- "Genetic Drift" is the name given to random fluctuations which result from the transfer of alleles from one generation to the next. This is particularly evident in geographically isolated populations (where what is called a "founder effect" often occurs — the coming to dominance of a particular trait due to repeated inbreeding). This can also occur in populations which have self-imposed isolation, or intense inbreeding due to social factors.

The result of genetic drift tends to be a reduction in the variation within the population, and an increase in the divergence between two populations which might originally have been similar.

- "Natural Selection" is what Charles Darwin originally proposed in his famous (and now often misquoted book whose full title is *On the Origin of Species by Means of Natural Selection, or the Preservation of Favoured Races in the Struggle for Life*). Natural selection means that the environment might change to a degree whereby an already existing DNA strand, or physical attribute, becomes dominant because the other DNA strands are not suited to survival in the changed environment.

The sickle cell example serves well to illustrate this point once again: Individuals who have the mutation for sickle cell appear to be more resistant to malaria than non-carriers so non-carriers were more often killed by that disease. The demise of the non-carriers of the mutation inevitably resulted in carriers becoming more numerous. Individuals of West African descent therefore make up the largest numbers of sickle-cell disease carriers.

- "Migration" means exactly that: the movement of different groups to other geographic regions. The movement of groups with different DNA composition inevitably brings about a wider genetic diversity in the territory to which they have moved (presuming, of course, that the area was already populated by another group). This is the reason for the existence of "mixed" groups in many regions of the globe.

- "Non-Random Mating Patterns" can lead to what is known as "inbreeding." This can also cause the dominance of certain traits, or, negatively, the creation of serious illness causing mutations. Perhaps one of the best-known examples of non-random mating patterns is that of the Jews.

That group's deliberate attempts to keep within their small founding group has led to the creation of a large number of genetic diseases among Jews, the most famous of which is Tay-Sachs Disease.

Short Tandem Repeats (STR) And Polymerase Chain Reaction (PCR) Testing

Short Tandem Repeats, or STRs, are short repetitive DNA sequences which are made up of usually no more than 13 nucleotides.

The number of times these STRs appears is determined by a process known *as polymerase chain reaction* (PCR) testing. This is a scientific process which allows researchers to make millions of copies of a complete or partial DNA sample so that it can be studied in detail.

PCR testing is most often used in criminal forensics and commercial ancestry testing.

Both of these processes — of course — have origin implications, in that they are used to determine the physical and other characteristics of an individual from a small sample. These characteristics include sex, hair color, skin color, eye color, and other attributes which clearly indicate the race, or physical appearance of the subject.

PCR testing allows law enforcement authorities, for example, to determine the physical appearance of suspects from a DNA sample alone. This physical appearance includes all the physical attributes which are popularly used to identify a person's "race" — this includes skin color, eye color, and so on.

PCR testing from STRs even allows for the reconstruction of specific facial characteristics. This process has already been used to help identify dead people and recreate the faces of historical personages.

How Ancestry and Origin are Determined

Genetic data retrieved from three sources: Y haplogroups; mtDNA haplogroups; and information from *autosomal DNA* (and specifically the alleles and SNPs); makes it possible to construct an accurate impression of:

- the geographic origin of that individual; and

– the physical appearance of that individual; and

– the characteristics of that individual (personality, cognitive ability, etc.)

This is in fact how the commercially available "home ancestry testing kits" work: by analyzing these three data sets, the origin, character, and appearance of an individual can be accurately mapped out.

Race is the Product of Variations in Alleles and SNPS

From the above overview of human DNA, it can be concluded that all humans share this basic structure. The question then arises: how are races, that is, groups of individuals with common physical and psychological characteristics, formed?

The answer to this question is surprisingly simple: it is the variation in genes which gives rise to the differences seen as race.

A gene (which, it will be recalled, is merely a collection of *nucleotides* on any given chromosome's DNA strand) which codes for light skin will be found in people with light skins. Conversely, a gene which codes for dark skin will be found in people with dark skins.

Similarly, genes which code for specific eye colors, hair colors, hair textures, skeletal structures (which includes skull shapes, bone densities, and bone lengths), and all of the other attributes by which race is identified, will only be found in the individuals, and groups of individuals, who display those specific traits.

These genes are always the result of specific arrays of alleles and specific SNPs. It is these DNA components which determine the outward "expression" of human genetics; in other words, how individuals are quite literally shaped and created.

When this "expression" takes the form of the physical appearance of a trait, the result is known as a "phenotype."

When large numbers of individuals share similar "gene expression" (that is, alleles and SNPs), then they tend to take on

a uniform appearance. This "uniform appearance" takes on the physical form of race.

To put it another way: it is not the similarity in human genetic structure which determines race: it is the way individual genes (alleles and SNPs) are constructed. These physical attributes — which are the result of "gene expression" — along with haplogroup categories, provide the genetic basis of all racial groups.

In the following chapters, examples will be provided of specific alleles, SNPs, and haplogroups which determine these physical characteristics.

In addition, the genes which code for cognitive ability — intelligence — and character will also be discussed.

CHAPTER THREE

RACE DEFINED BY Y AND mtDNA HAPLOTYPE GROUPS

The reader will have noted from the previous chapter that there are two sorts of DNA which are always passed down unchanged from father to son, and mother to daughter. These are Y-Chromosomes for males, and mtDNA for females.

The reader will also have noted that physical characteristics, which manifest themselves as outwardly visible racial characteristics, are determined by individual "genes"—alleles and single-nucleotide polymorphisms (SNPs).

Y and mtDNA Chromosomes Provide Geographically Differentiated Human Origin Trees

The Y and mtDNA categories—their haplogroups and subclades—therefore provide the primary "ancestry trees" of human differentiation, and the "genes" provide the actual physical manifestation, or "expression" of race.

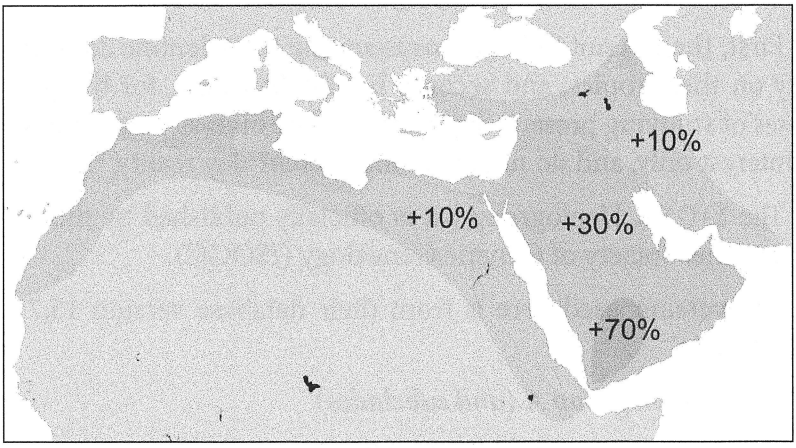
As race is a combination of both of these factors, it is possible for an individual to have the Y-Chromosome or mtDNA haplogroups of one "ancestry tree" and some or all of the physical attributes of another group: this is the result of mixing.

In other words, where an individual has the Y-chromosome haplogroup or mtDNA haplogroup of one ancestry tree, and some or all of the physical characteristics associated with another race, then it means that that individual is of mixed descent: at some stage in their family tree, a male or a female from another group has bred into that line.

Some practical examples of this process at work will be given later.

Nonetheless, by studying the geographic spread of Y-chromosome and mtDNA haplogroups, it is possible to determine the geographic region where that haplogroup originated, and even to venture fairly accurate estimates of how long ago this happened.

An example of this is given in the illustration below: the Y-Haplogroup J1 (which is a subclade of the Y-Haplogroup J) is concentrated in the Arabian Peninsula and its immediate surrounding areas.



Distribution of the J1 Y-Haplogroup

It is therefore clear that the Y-Chromosome J1 is a "Semitic" haplogroup, associated with individuals who have Semitic ancestry.

Another few examples will cement the point: Y-Haplogroups A00, A0-P305, A1a-M31, A1b-M6, or B-M60, will all indicate African ancestry. The subclades can even be broken down into regions: for example, the A00 subclade is linked to the Bangwa tribe in the country of Cameroon, and the A1b-M6 haplogroup with the Khoisan tribe (formerly called "Bushmen"), the pre-black African peoples of southern Africa.

This sort of ancestry determination has been frequently used by those of African descent in the Americas attempting to deter-

mine their original roots in Africa. Y and mtDNA Haplogroups *therefore serve as race separators.*

It is important to note, once again, that they represent genetic patterns found in certain populations, but do not, by themselves, make any definite presumptions about the phenotype, or physical appearance, of those individuals.

Y-Chromosome Family Trees by Geographic Origin

This overview will not concern itself with the ongoing debates over (1) the age of the various haplogroups, or (2) the age of claimed divergences in the haplogroup trees.

First, there is not full consensus among the scientific community on these topics; and second, both debates are, for the purposes of studying present-day racial genetic diversity, of academic interest only, and do not affect the present-day reality.

The Y-DNA Haplogroup list is officially published by the International Society of Genetic Genealogy (ISOGG).

The version used here is from their database version 15.73, July 2020.

- ***Y-DNA Haplogroup A (and subclades)***

  Geographic concentration: Sub-Saharan Africa.

  Y-Haplogroup A subclades A1-P305 are found in in Central and West Africa; A1a-M31 in northwestern Africans; A1b1a-M14 is found among the Khoisans—the original pre-Bantu inhabitants of southern Africa (previously called "Bushmen")—and certain east African populations.

- ***Y-DNA Haplogroup B (and subclades)***

  Geographic concentration: Sub-Saharan Africa.

  Haplogroup B is found in higher frequencies in northeastern Africa and in the Khoi-San. It is also found in present-day populations in the rest of sub-Saharan Africa, including the Pygmies.

- *Y-DNA Haplogroup C (and subclades)*

  Geographic concentration: East Asia (Japan, China, Korea, subclade M217); Oceania (Australian Aborigines, subclade C1).

  Traces of this haplogroup and its subclades are also widely dispersed in smaller amounts around Southeast Asia, the Maoris in New Zealand, in Central Asia, parts of Eastern Europe, and in Amerindian populations in North America. A major subclade is found among the Aborigines in Australia and is usually categorized separately.

- *Y-DNA Haplogroup D (and subclades).*

  Geographic concentration: East Asia.

  Most commonly found in Tibet (at a range of 50 percent) and in Japan (35 percent), this Y-haplogroup and its subclades can be found as far afield as the Asiatic influenced populations of Russia, Nepal, Kazakhstan, and Korea. It is also found in lower frequencies in India, the Philippines, and, albeit it in very low quantities, in Nigeria.

- *Y-DNA Haplogroup E (and subclades).*

  Geographic concentration: Central Africa.

  Concentrated in north central Africa, Y-haplogroup E and its major subclades—E1b1a and E1b1b—are the single most common haplogroups in Africa. Subclade E1b1b evidently moved out of northern Africa in antiquity, possibly due to mixing in early Egyptian and during the era of the Classical Civilizations.

  As a result, traces can be found spread around the Mediterranean Basin, and in other European regions. It is also found in Ashkenazi Jews, and in the Middle East, including the Arabian Peninsula.

- *Y-DNA Haplogroup F (and subclades).*

  Geographic concentration: Central Asia.

This haplogroup has been found anywhere else apart from in Sri Lanka, China, India, and Vietnam.

- ***Y-DNA Haplogroup G (M201) (and subclades).***

  Geographic concentration: Central Asia.

  Found in low frequencies in parts of Europe, South Asia, Central Asia, and North Africa. Common to the people of the Caucasus.

- ***Y-DNA Haplogroup H (and subclades).***

  Geographic concentration: Central Asia (Indian subcontinent).

  The Romani (previously known as "Gypsy") population in Europe is also marked by the Y-DNA Haplogroup H. The Romani are not to be confused with what many in Western Europe call the "traveler community," often mistakenly called "Gypsies" as well. The latter group are often of Irish or European ancestry and have nothing do with India.

- ***Y-DNA Haplogroup I (and subclades).***

  Geographic concentration: Europe.

  Y-DNA haplogroup I is unique to Viking or Scandinavian origin males and appears almost nowhere outside of Europe. Subclade I2a2a1-M284 occurs almost exclusively in Britain.

- ***Y-DNA Haplogroup J (and subclades).***

  Geographic concentration: Middle East.

  This haplogroup is concentrated on the Arabian Peninsula, and radiates out through North Africa, Europe, Central Asia, Pakistan, and India. Haplogroup J is also dominant in Ashkenazi and Sephardic Jews, in particular subclades J2 and J1 respectively.

- ***Y-DNA Haplogroup K (and subclades).***

  Geographic concentration: Central Asia.

Found in Bali, Indonesia, India, Pakistan. Also found in Australian Aborigines and other islands in the region.

- ***Y-DNA Haplogroup L (and subclades).***

  Geographic concentration: Central Asia.

  This haplogroup is concentrated in Pakistan, India, Sri Lanka, and Bangladesh. It has also dissipated out as far away as Iraq, Bahrain, Kuwait, Saudi Arabia, and southern Europe.

- ***Y-DNA Haplogroup M (and subclades).***

  Geographic concentration: Oceania.

  This haplogroup is most common in Papua New Guinea, where it is two-thirds of the population. Some traces can also be found among the Australian Aborigines.

- ***Y-DNA Haplogroup N (and subclades).***

  Geographic Concentration: Arctic Circle (East Asia).

  This haplogroup is marked among the Saami, Siberian Eskimos, and other populations of the far north of Europe and Russia.

- ***Y-DNA Haplogroup O (and subclades).***

  Geographic concentration: East Asia.

  Highest frequencies in China, Japan, and Korea. Also found in Indonesia, Melanesia, Micronesia, and Polynesia.

- ***Y-DNA Haplogroup P (and subclades).***

  Geographic concentration: East Asia.

  Notes: Found in the Philippines, India, China, and other closely related regions.

- ***Y-DNA Haplogroup Q (and subclades).***

  Geographic concentration: Americas.

  The Y-DNA Q-M3 subclade moved with early migrations into the Americas via northern Eurasia and is today almost exclu-

sively associated with North, Central, and South American Indian populations. Other subclades are found in Vietnam, Siberia, Indonesia, Bhutan, Uzbekistan, Pakistan, the Middle East, and small numbers of variants in Europe.

- ***Y-DNA Haplogroup R (and subclades).***

  Geographic concentration: Europe.

  Subclades R1, R1a, and R1b are the most common Y-DNA haplogroups in Europe. R1a is believed to have entered Europe along with Indo-Europeans around 5,000 years ago, while R1b is believed to have entered Europe several thousand years earlier as part of a migration from the Steppes.

  R1b subclades reach into northern Africa and southwest Asia and are associated with male admixture in historical times. The appearance of Y-DNA haplotypes in India is associated with Indo-European incursions around 4,000 years ago.

- ***Y-DNA Haplogroup S (and subclades).***

  Geographical concentration: Oceania.

  A distinctive Y-haplogroup from Papua New Guinea which is also found in the adjacent islands (Indonesia, Melanesia, and the Philippines). Also found in Australian Aborigines.

- ***Y-DNA Haplogroup T (and subclades).***

  Geographic concentration: Middle East.

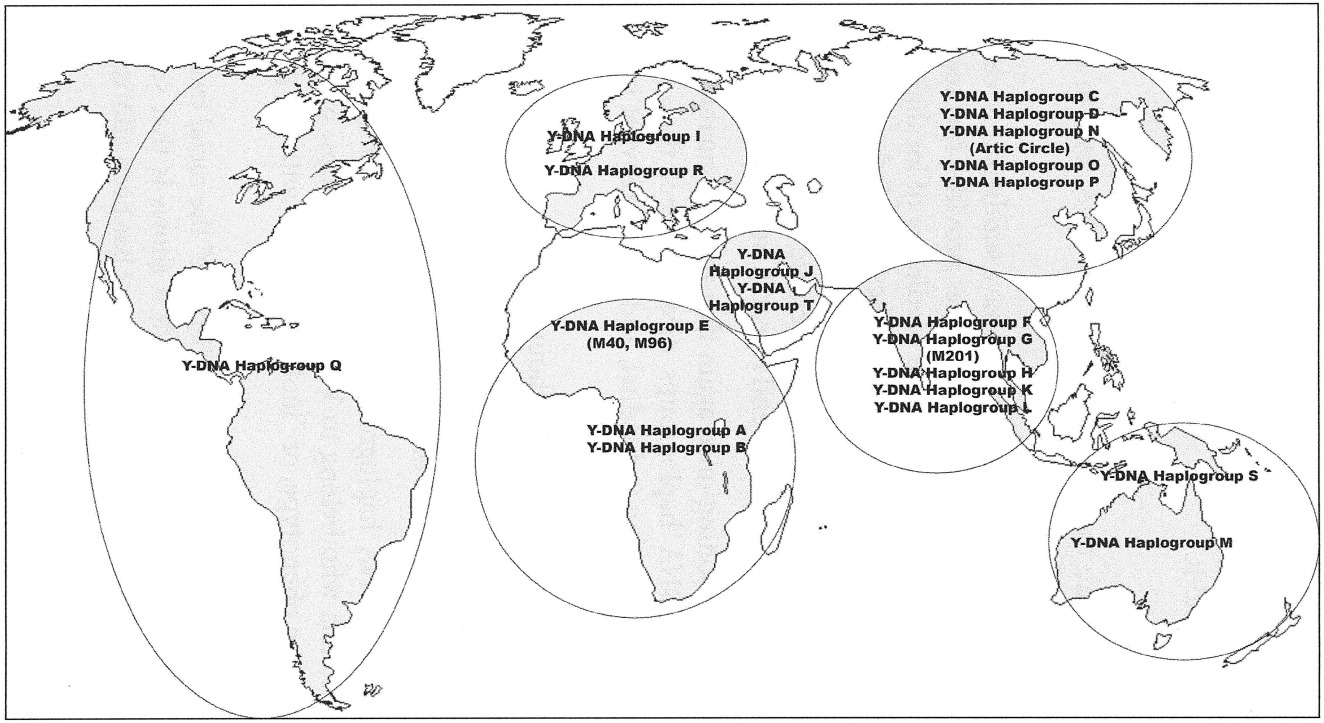
  A rare haplotype, found in the Middle East, and in small amounts in the northern Mediterranean Coast and Europe.

Y Haplotypes Divide Humans into Seven Major Geographical Categories

From the above list, the reader will have observed that there are seven major geographic regions for the Y-haplogroups.

1. Africa (Sub-Saharan): A, B, and E (M40, M96).

2. East Asia: C (M217), D, N, O, and P.



The major Y-DNA haplogroups as found on the globe in their originating positions. Each major haplogroup has several divisions, called "subclades" which are too numerous to list on this map.

3. Central Asia: F, G (M201), H, K, and L.

4. Middle East: J, and T.

5. Oceania: M, S, and C1.

6. Europe: I, and R.

7. Americas: Q.

These regions are the same ones used in DNA tests to help determine "ancestry."

All these major Y-DNA haplogroups have subdivisions, called subclades. A breakdown of these subclades provides further insight into how they serve as accurate predictors of localized regions. There are a very large number of these subclades—far too many to reprint here—but which can be easily looked up on the website of the International Society of Genetic Genealogy, which is regularly updated as new data and research becomes available.[1]

It is worth mentioning once again that there is currently no evidence that Y-DNA haplogroups directly affect the overall physical appearance of an individual, and therefore what is perceived as a "race."

Y-DNA groups do, however, without question, indicate paternal origin from specific areas. Those areas are in turn associated with specific "looks," or "phenotypes," and Y-DNA does therefore serve as a valuable indicator of an individual's "racial tree."

A study of Y-DNA haplogroups also reveals that there are many individuals who belong to Y-DNA haplogroups, but who do not share the phenotype, or "look," associated with the geography of their haplogroup.

This is not a mystery, nor does it "disprove" the racial tree origin provided by haplotype analysis. All it shows is that somewhere in that individual's paternal line, there has been admixture at some stage, ancient or recent.

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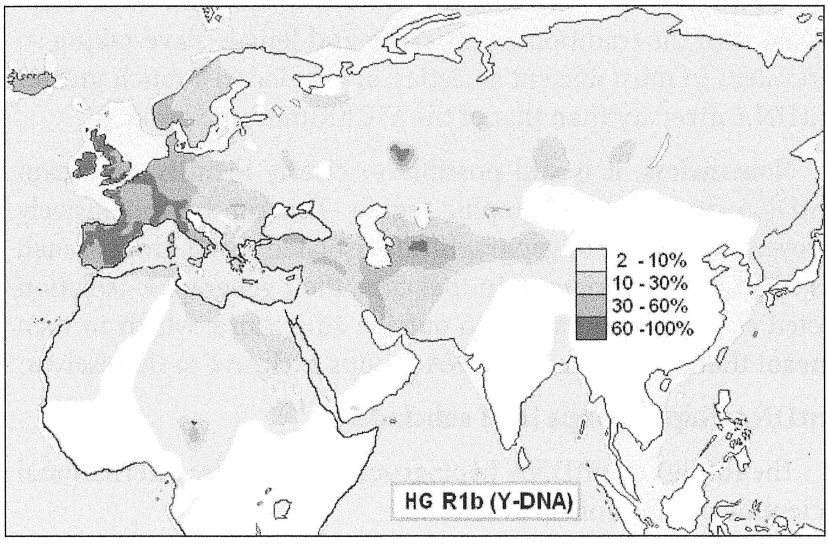
1 See isogg.org/tree/index.html (retrieved October 2022).

For example, a detailed map of the distribution of the R1b Haplogroup—a European haplogroup—shows that its frequencies are highest in western Europe, and the frequency of occurrence radiates out from there. This wider dispersion means only that males carrying the R1b Haplotype have, at some stage, admixed with individuals from other regions.



The distribution of the R1b-Y chromosome haplogroup show how far admixture has spread out from Europe.

An individual from one of the central African states, for example (where R1b can be found in very low frequencies), will likely show the *physical characteristics associated with Africans*, despite having a European paternal haplogroup.

This is, as mentioned earlier and explained in detail in the next chapter, because physical appearance is primarily determined by a separate set of genes.

mtDNA-Chromosome Family Trees by Geographic Origin

In the same way as Y-Chromosomes represent paternal racial "family trees," mitochondrial DNA (mtDNA) represents the corresponding female descended family trees. There are two major differences between Y-chromosome family trees and mtDNA

family trees:

(1) There are a larger number of mtDNA haplogroups; and

(2) They tend to be more widespread because in most societies there was always a higher female than male migration rate (via "patrilocality," the tendency for a wife to move into her husband's household, instead of the other way around). This, combined with the traditional patriarchy and female slave-taking so prevalent in most ancient societies, always led to a much greater mtDNA diversity than that of the Y-Chromosome groups.

Nonetheless, it is still possible to clearly identify dominant mtDNA groups by geographic region. This can be conclusively proven in the case of geographically isolated racial groups, such as Australian Aborigines. In that case, their geographic isolation acted as a physical barrier to outside admixture, which in turn meant that they possess mtDNA groups exclusive to themselves.

mtDNA Haplogroups (and subclades)

The full list of mtDNA haplogroups, as used by international scientific convention, is as follows:

· ***mtDNA Haplogroup A (and subclades).***

Geographic concentration: Americas.

Highest frequencies among Amerindians. A high prevalence in East Asia indicates the ultimate origin of the American Indian population.

· ***mtDNA Haplogroup B (and subclades).***

Geographic concentration: East Asia.

Highest in China, also prevalent in Amerinds.

· ***mtDNA Haplogroup C (and subclades).***

Geographic concentration: East Asia.

Highest in Arctic Circle region, also prevalent in Amerinds.

· ***mtDNA Haplogroup D (and subclades).***

Geographic concentration: East Asia.

Highest concentration in northeast Russia and China, also prevalent in Amerinds.

· ***mtDNA Haplogroup E (and subclades).***

Geographic concentration: East Asia.

Most prevalent in the Malay Archipelago.

· ***mtDNA Haplogroup F (and subclades).***

Geographic concentration: East Asia.

Found in China, and the nations of Indochina (Thailand and Vietnam).

· ***mtDNA Haplogroup G (and subclades).***

Geographic concentration: East Asia.

Found in Japan (particularly among the Ainu people), Mongolia, and Tibet. Small frequencies in other regions of East Asia. Completely absent in Amerindians.

· ***mtDNA Haplogroup H (and subclades).***

Geographic concentration: Europe.

The single most common European haplogroup.

Subclade H8 is found in Central Asia and is likely linked to admixture with pre-Indo-European populations.

· ***mtDNA Haplogroup I (and subclades).***

Geographic concentration: East Africa.

Also found in low frequencies in West and South Asia, and southern Europe.

· ***mtDNA Haplogroup J (and subclades).***

Geographic concentration: Middle East.

Found in lower frequencies in Europe, north Africa, India, and isolated regions in Sub-Saharan Africa.

· ***mtDNA Haplogroup K (and subclades).***

Geographic concentration: Europe.

One of the rarer haplogroups found in Europe. Subclades K1a1b1a, K1a9, and K2a2a are often found in Ashkenazi Jews. It is also common in some Middle Eastern populations, particularly among Kurds, indicating an Indo-European origin at some stage.

· ***mtDNA Haplogroup L (and subclades).***

Geographic concentration: Africa, Sub-Saharan.

A uniquely sub-Saharan mtDNA haplotype, with its subclades L0 – L6 dominating the continent. Found in small frequencies in central West Asia and Europe, dating from early admixture, and in America following the trans-Atlantic slave trade era.

· ***mtDNA Haplogroup M (and subclades).***

Geographic concentration: East and Central Asia.

mtDNA Haplogroup M and its subclades are the most common haplogroup in Asia, accounting for 60 percent of all female lineages.

· ***mtDNA Haplogroup N (and subclades).***

Geographic concentration: Central and East Asia.

Most mtDNA N subclades are found in East Asia, with declining frequency moving west.

Smaller frequencies of other mtDNA N subclades are found in the Arabian Peninsula, the Middle East, North Africa, and isolated parts of south Russia (Caucasus region).

· ***mtDNA Haplogroup O (and subclades).***

Geographic concentration: Oceania (Australia).

This mtDNA haplogroup is ultra-rare, even in Australian Aborigines, where only a tiny number have been detected.

· ***mtDNA Haplogroup P (and subclades).***

Geographic concentration: Oceania.

Highest frequencies in Australian Aborigines, then in the islands to the north of Australia, including Papua New Guinea and Melanesia.

· *mtDNA Haplogroup Q (and subclades).*

Geographic concentration: Oceania.

Highest frequencies in islands off Australia and the South Pacific.

Also found among Australian Aborigines.

· *mtDNA Haplogroup R:*

A so-called "super group" with important and differentiated geographic subclades.

- *mtDNA Haplogroup R0a (and subclades).*

Concentrated in the Arabian Peninsula and found in small frequencies in the Middle East.

- *mtDNA Haplogroup R1 (and subclades).*

Low frequencies mainly in Brahmin (upper caste) Indians. Also found in isolated Arctic Circle tribes of Asiatic origin.

- *mtDNA Haplogroup R2 (and subclades).*

Concentrated in Pakistan, India, and found in low frequencies in Iran, Georgia, and Turkey.

- *mtDNA Haplogroup R3.*

Concentrated in Armenia.

- *mtDNA Haplogroups R5, R6, R7, R8, R30, R31 (and subclades).*

Found in India at high frequencies.

- *mtDNA Haplogroup R9 (and subclades).*

Found in Indonesia, Indochina, Malaysia (only in Aboriginal Malays).

- ***mtDNA Haplogroup R11 (and subclades).***

  Highest frequencies in China. Subclades found in lower frequencies in Japan, Korea, and Indochina.

- ***mtDNA Haplogroup R12, R14, R22, R23.***

  Highest frequencies in Australian Aborigines, subclades in East Asia, and notably in Polynesia.

- ***mtDNA Haplogroup R21.***

  Concentrated in Negroid ("Negrito") population of Malaysia.

· ***mtDNA Haplogroup S (and subclades).***

  Geographic concentration: Oceania.

  Found in Australian Aborigines.

· ***mtDNA Haplogroup T (and subclades).***

  Geographic concentration: Europe.

  Highest frequency in central Europe.

  Also found in the Middle East, centered between the Black and Caspian seas, likely from the Indo-European era.

· ***mtDNA Haplogroup U: A so-called "super group" with important and differentiated subclades.***

- ***mtDNA Haplogroup U1 (and subclades).***

  Low frequency in Eastern Europe. Also found in Central Asia and India.

- ***mtDNA Haplogroup U5 (and subclades).***

  Mainly concentrated in the pre-Indo-European population of Europe.

- ***mtDNA Haplogroup U4.***

  Found in very low frequencies in Europe and limited to the Arctic Circle populations with Asiatic origin such as the Saami (formerly called the "Lapps").

- *mtDNA Haplogroup U8.*

  Most common among the Basque population, indicating that it is an ancient European haplotype which predates the Indo-European era.

- *mtDNA Haplogroup U6 (and subclades).*

  Likely a remnant of historical migrations to the area from Europe or the near Middle East.

- *mtDNA Haplogroup U3.*

  Found among the Berbers in North Africa, and in Egypt.

- *mtDNA Haplogroup U6 (and subclades).*

  Concentrated in India.

- *mtDNA Haplogroup U9.*

  Limited frequency in Pakistan. Also found in limited frequencies in East Africa, with the two occurrences possibly linked to the Muslim slave trading era.

- *mtDNA Haplogroup U7.*

  Highest concentration among Saudi Arabians. Also found in the Middle East and parts of India.

· *mtDNA Haplogroup V.*

  Geographic concentration: Arctic Circle (Asiatic origin, Northern Europe, Saami).

  mtDNA Haplogroup V is Asiatic (Siberian) in origin and is found at high levels in the Saami (previously called the "Lapp") people in the far north of Europe. Also found in lower frequencies in northwest Africa. Although widely claimed as a "European" haplogroup, it is almost completely absent in European racial groups.

· *mtDNA Haplogroup W.*

  Geographic concentration: Central Asia.

The highest concentration is found in northern Pakistan. It is also found in West Asia, the Middle East, and Europe, which likely indicates a link to the movement of Indo-European tribes through the region.

- ***mtDNA Haplogroup X.***

  Geographic concentration: America.

  Found in 13 percent of Amerinds. Also found in the Middle East, the southeast Balkans, and subclades at low frequencies in Europe, north Africa, the Arabian Peninsula, and West Asia.

  It likely entered the Americas at the time of the populating of that continent by the Asiatic tribes who became the Amerinds.

- ***mtDNA Haplogroup Y.***

  Geographic concentration: East Asia.

  Found in high frequency in the Asiatic origin population in the far northeast of Russia, close to the sea border with Alaska. Subclades found in isolated parts of Indonesia.

- ***mtDNA Haplogroup Z***

  Geographic concentration: East Asia.

Found in highest concentration in Arctic Circle Asiatic origin peoples of central north Russia.

mtDNA Haplogroups Divide Humans into Seven Major Geographical Categories

From this list, the reader will have observed—just as is the case with Y chromosomes—that there are seven major geographic regions for the mtDNA haplogroups.

1. Africa (Sub-Saharan): I, L, U3, and U6.

2. East Asia: B, C, D, E, F, G, R11, Y, and Z.

3. Central Asia: M, R1— R9, R30, R31, U6, U9, V, and W.

4. Middle East: J, N, R0a, and U7

5. Oceania: O, P, Q, R12, R14, R21, R22, R23, and S.

6. Europe: H, K, T, U1, U5, and U8.

7. Americas: A, B, C, D, and X.

The evidence from both the Y chromosome and mtDNA chromosome haplogroups is clear: they both indicate that there are seven major geographical regions of the earth which have traditionally been inhabited by groups of people sharing a common haplotype inheritance.

Put in graphical form, these mtDNA haplogroups appear as a map on the next page.

Causes of Haplogroup Admixture

Over the course of history—from ancient times to the present—there has been a certain amount of admixture, which has caused some of these haplotypes to be transferred to other regions.

This accounts especially for the wider distribution of mtDNA haplogroups.

Proven historical migrations—as opposed to those still under debate, such as the "single-origin theory" versus the "multi-regional origin theory" of human origins—have also played a role in distributing Y and mtDNA haplotypes.

For example, the spread of the Indo-European language speakers from the Steppes (and the region around the Black Sea) around 5,000 BC impacted a region stretching from present-day Pakistan to Ireland, and from Scandinavia to Egypt.

Then, the repeated invasions of Europe by the Huns and other Asiatic tribes, followed by the Muslim expansion from Saudi Arabia into the Middle East, North Africa, and finally into Europe (Spain, Portugal, and southern France in the west; and into the Balkans as far as Vienna in the east), all also clearly had an impact upon the distribution of haplogroup types.

The colonial era, which saw Europeans spread out over the earth and settle new regions, also brought about a mass migration of haplogroups (mostly European) to the Americas, Oceania, and elsewhere.

Despite all these historical processes, the genetic lines remain clear. It is in fact a tribute to present-day science that, through all the vagaries of history, it is still possible to identify specific haplogroups and their regions.

Once again, all of this ultimately proves that race is not a "social construct," but rather has a firm base in biology. That biological evidence clearly shows that there are seven major groups of people, or seven major races.

With this firmly established, all that remains is to see how DNA physically shapes these different races so that their differences are visible to the naked eye.

The major mtDNA haplogroups as found on the globe in their originating positions. Each major haplogroup has several divisions, called "subclades" which are too numerous to list on this map.

CHAPTER FOUR

RACE DEFINED BY AUTOSOMAL DNA

The previous chapter provided an insight into how it is possible to classify individuals according to their inherited Y and mtDNA chromosome haplotypes. This is, however, only one part of the process in which DNA shapes the seven major races of the world.

Autosomal DNA, the chromosomes which are not sex specific, play an equally important—if not perhaps a dominating role—in shaping the physical appearance of racial groups.

The Role of Single Nucleotide Polymorphisms (SNPs) in Shaping Racial Differences

This is because autosomal DNA, specifically the Single Nucleotide Polymorphisms (SNPs) components (including the exons and introns),[1] "codes" or makes the unique protein chains which in turn determine how the genes are "expressed." By "expressed" is meant how these genes manifest themselves in the human body: how they determine, for example, eye color, hair color, skeletal structure, and so on. In other words, it is the way in which the SNPs are configured which determines the functions of the various genes.

It is the difference in these genes—or, more directly put—the difference in the way that the SNPs (and its components) are configured—which cause racial characteristics. There are many genetic studies which have conclusively determined the role of

1 See Chapter Two for a refresher on this structure.

SNPs in determining race, but perhaps the most significant one to date was produced by an international team of scientists in 2010.

That study, titled "Toward a more uniform sampling of human genetic diversity: A survey of worldwide populations by high-density genotyping,"[2] was prepared in a joint effort by ten scientists from the University of Utah, one from the Molecular Genealogy Foundation in Utah, and three from the University of Toronto, Canada.

Published in the journal *Genomics* (Volume 96, Issue 4, October 2010, Pages 199–210), this study conducted its own DNA sampling from 13 worldwide populations "not covered by previous studies" and then combined them with already established and verified worldwide samples consisting of at least 250,000 SNPs from 40 populations spread around the globe on all continents.[3]

The sheer scale of the study makes it one of the most important such overviews conducted to date, not only for ancestry determination, but also, as the paper said, for the evaluation of "genetic variation responsible for disease" for which the science is "providing a foundation for important biomedical studies."[4]

The scientists used what is known as "Affymetrix Microarray Technology," which is a complicated chemical process that allows an insight into the "expression profile" (that is, how the SNPs appear) of literally thousands of genes within a single experiment.

The samples developed were then compared with each other to determine similarities or differences between the SNPs. Using this technology, it becomes possible to map just how close—or how far away—SNPs are from each other in terms of their innate composition. For the purposes of their study, the scientists

2 "Toward a more uniform sampling of human genetic diversity: A survey of worldwide populations by high-density genotyping," Xing, J., et al., *Genomics* 96 (2010), 199–210.
3 *Ibid.*
4 *Ibid,* page 199.

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Toward a more uniform sampling of human genetic diversity: A survey of worldwide populations by high-density genotyping

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ABSTRACT

High-throughput genotyping data are useful for making inferences about human evolutionary history. However, the populations sampled to date are unevenly distributed, and some areas (e.g., South and Central Asia) have rarely been sampled in large-scale studies. To assess human genetic variation more evenly, we sampled 296 individuals from 13 worldwide populations that are not covered by previous studies. By combining these samples with a data set from our laboratory and the HapMap II samples, we assembled a final dataset of ~250,000 SNPs in 850 individuals from 40 populations. With more uniform sampling, the estimate of global genetic differentiation (F_{ST}) substantially decreases from ~16% with the HapMap II samples to ~11%. A panel of copy number variations typed in the same populations shows patterns of diversity similar to the SNP data, with highest diversity in African populations. This unique sample collection also permits new inferences about human evolutionary history. The comparison of haplotype variation among populations supports a single out-of-Africa migration event and suggests that the founding population of Eurasia may have been relatively large but isolated from Africans for a period of time. We also found a substantial affinity between populations from central Asia (Kyrgyzstani and Mongolian Buryat) and America, suggesting a central Asian contribution to New World founder populations.

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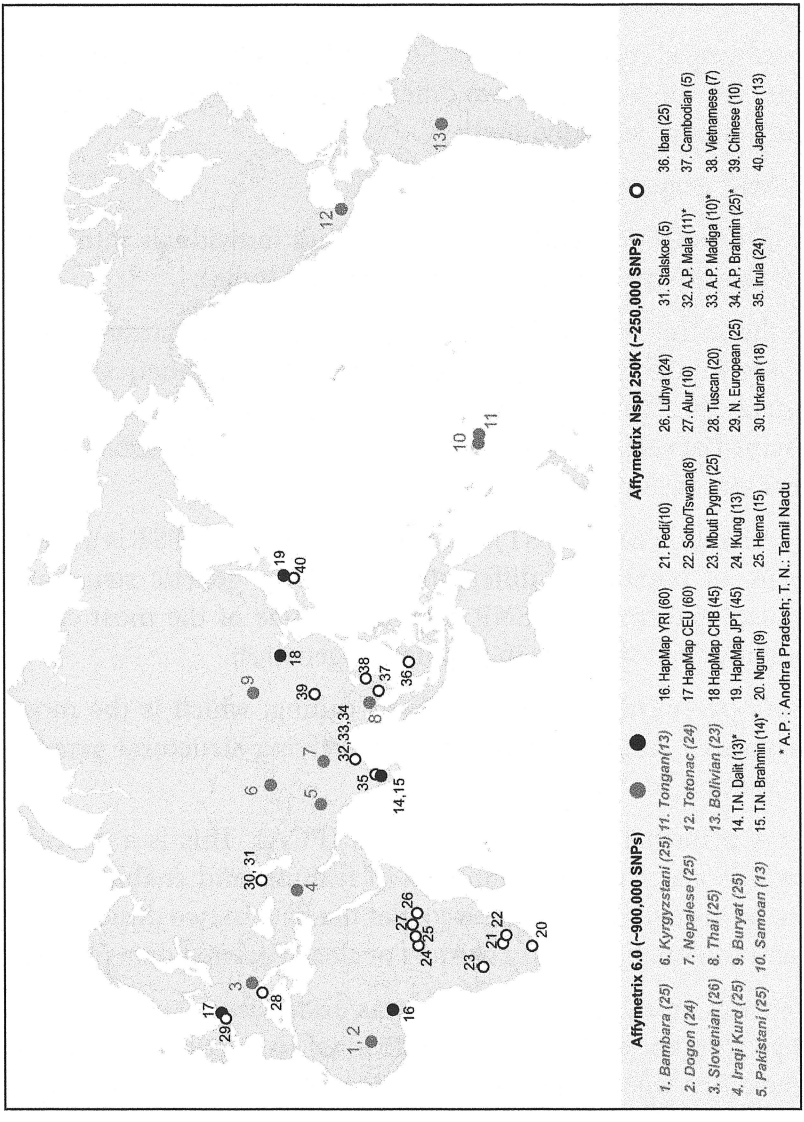
analyzed SNPs from populations from the following 40 regions, making sure to only collect from "native" or "indigenous" peoples in all cases. The widespread nature of the samples is demonstrated by the following map and its key:



This meant that samples were taken from 13 worldwide populations, specifically West Africa, Central Europe, West Asia, Central Asia, South Asia, Southeast Asia, Polynesia, and America (the reader will notice the absence of samples from Australia).

The raw data so extracted was then subjected to a series of complex analytical processes. These processes included:

- advanced comparisons for SNP and haplotype heterozygosities (the possession of two different alleles of a particular gene or genes by an individual);

- SNP genotyping;

- SNP selection and the division of all individuals into four major groups (Africa, Asia, Europe, and India);

- the testing of each SNP for deviations from the Hardy–Weinberg Equilibrium (a principal of population genetics which states that "genotype frequencies in a population remain constant between generations in the absence of disturbance by outside factors");

- A fixation index (FST) was then generated. An FST is a measure of population differentiation due to genetic structure, calculated from the SNP data, and is one of the most commonly used statistics in population genetics;

- Copy Number Variants (CNV) calling, which is the most common scientific method of identifying structural genetic variation; and

- A principal component analysis (PCA). This is a process which analyzes large numbers of features and enables them to be visualized on a graph to plot the data in two dimensions and to visually identify clusters of closely related data points.

Finally, an admixture analysis was undertaken "to determine the genetic ancestries of each individual in a given number of populations without using information about population designation."[5]

5 *Ibid,* page 202.

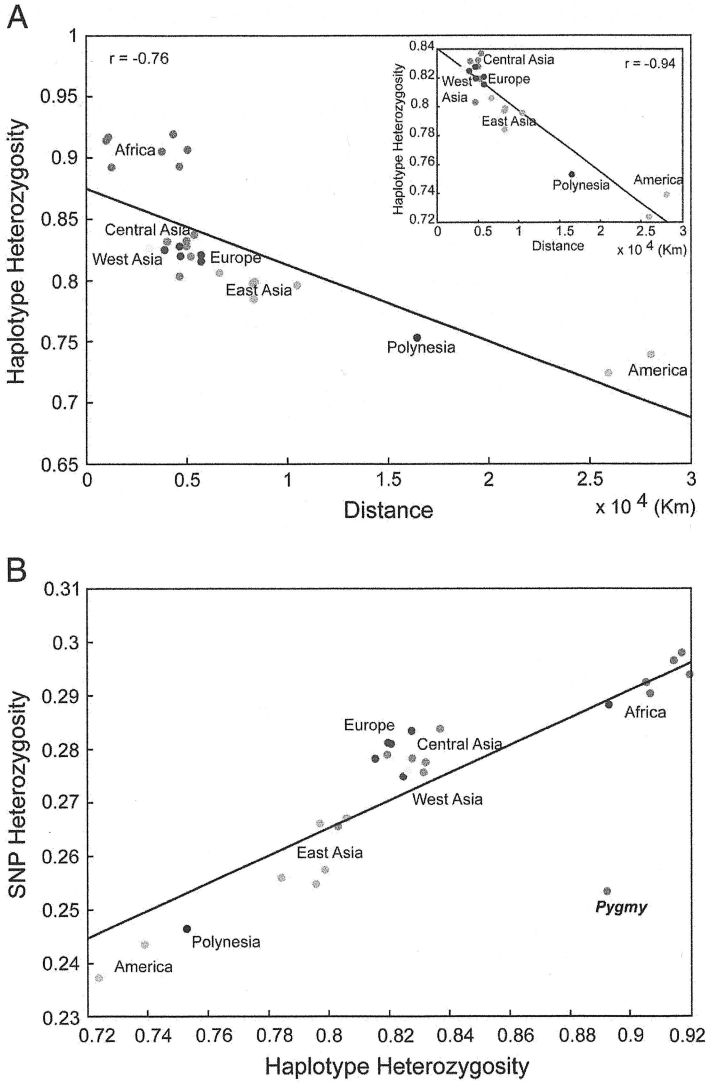


Figure 2 from the Genomics paper, "Toward a more uniform sampling of human genetic diversity: A survey of worldwide populations by high-density genotyping" (2010). Titled "SNP diversity," chart A presents "SNP haplotype diversity as a function of geographic distance from East Africa." The correlation improved substantially when African populations were excluded (as seen in the upper right panel). Chart B shows "SNP haplotype diversity versus SNP heterozygosity."

SNP Analysis Divides World's Population into Seven Distinct Categories

The results, when projected out in schematic format, were dramatic. As the study's figure 2 (previous page) showed, the genetic distance between the samples was marked and obvious.:

The most dramatic representation was, however, in the study's Figure 3b, which is a representation of the principal component analysis (PCA) conducted on the entire dataset.

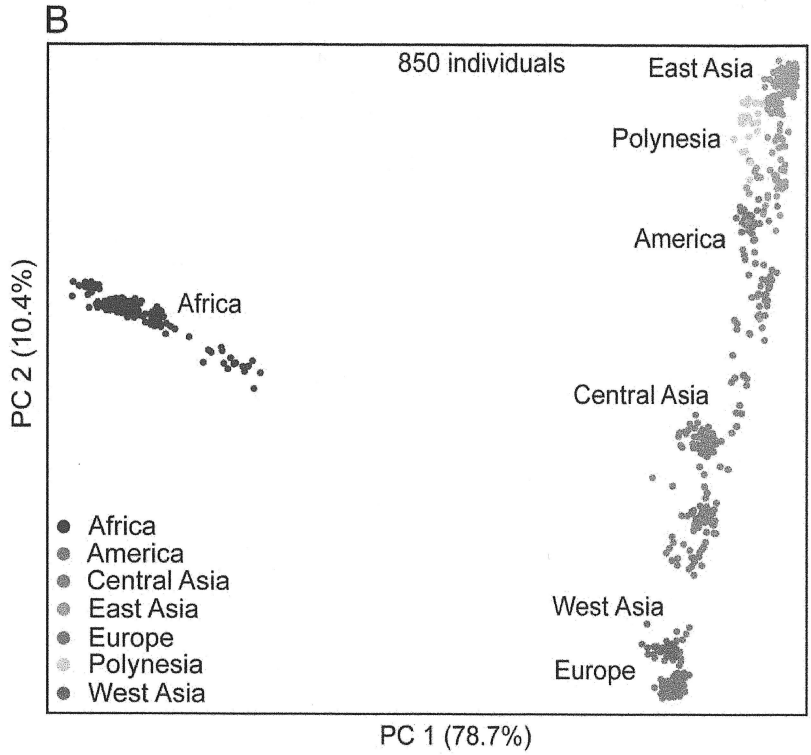


Figure 3b from the Genomics *paper, "Toward a more uniform sampling of human genetic diversity: A survey of worldwide populations by high-density genotyping" (2010). Titled "Principal component analysis. First two principal components (PCs) are shown. Each individual is represented by one dot and the label corresponding to their regional origin. The percentage of variance explained by each PC is shown on the axis."*

This analysis showed that the sample groups, as represented by their SNPs in autosomal data, differed from each other as widely as their geographic distances, and could easily be divided up into common groups, quite distinct from each other.[6]

The study then went on to describe the divisions as "Africa, America, Central Asia, Europe, Polynesia, and West Asia." This is, needless to say, nearly identical to the Y and mtDNA haplogroup divisions, with the minor exception that some datasets are missing from the global SNP analysis (such as Australian Aborigines and samples from the Arabian Peninsula) and are therefore not represented in the final summary.

The missing data from Australian Aborigines was included in another, similar study, published in the journal *Nature*. That paper, titled "Genotype, haplotype and copy-number variation in worldwide human populations,"[7] provided an analysis of "high-quality genotypes at 525,910 single-nucleotide polymorphisms (SNPs) and 396 copy-number-variable loci in a worldwide sample of 29 populations."[8]

The analysis showed that the Australian Aborigine SNP samples placed that race firmly into the "Oceania" category, once

6 The great distance between the African sample and the rest of the human types, as so vividly illustrated in *Figure 3b* above, has been the source of much debate. The most likely reason for the huge gap between African SNP samples and other races is the existence of what is known as "ghost DNA" in Africans. This African "ghost DNA," was first brought to the scientific world's attention in 2020, when a paper published in the journal *Science Advances* ("Recovering signals of ghost archaic introgression in African populations," Durvasula, A., et al., *Science Advances*, Feb. 12, 2020, Vol 6, Issue 7) announced that around 19 percent of DNA sequences in West Africa come from no known human group. One of the paper's authors, in an interview with National Public Radio (NPR), said that he thinks "it comes from a yet-to-be-discovered group . . . We don't have a clear identity for this archaic group. That's why we use the term 'ghost.' It doesn't seem to be particularly closely related to the groups from which we have genome sequences from" ("'Ghost' DNA In West Africans Complicates Story of Human Origins," NPR, February 12, 2020).

7 "Genotype, haplotype, and copy number variation in worldwide human populations," Jakobsson, M. et al., *Nature*, Feb 21, 2008; 451(7181):998–1003.

8 *Ibid*, page 998.

again confirming the genetic basis for seven major racial divisions on earth.

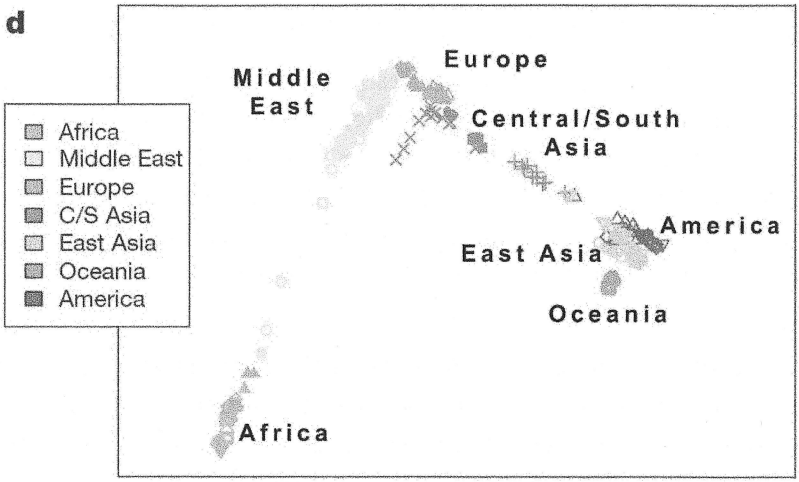


Figure 1d from the Nature paper, "Genotype, haplotype, and copy-number variation in worldwide human populations" (2008), which included datasets from Australian Aborigines in autosomal SNP analysis.

This multidimensional scaling (MDS) illustration demonstrates SNP, haplotype, and copy number variants (CNV) across the seven major racial categories.

Yet another scientific paper, "Haplotype variation and genotype imputation in African populations," published in the journal *Genetic Epidemiology* (2011),[9] which was focused on determining the genetic makeup of the African continent, provided a graphic which had as its purpose the demonstration of cross gene flows between Africa and elsewhere.

Using SNP analysis alone, the authors of that paper also came to the conclusion that there were seven major races on earth, and depicted this finding in a dramatic block scheme as follows:

9 "Haplotype variation and genotype imputation in African populations," Huang, L., et al., *Genetic Epidemiology,* Volume 35, Issue 8, December 2011, Pages 766–780.

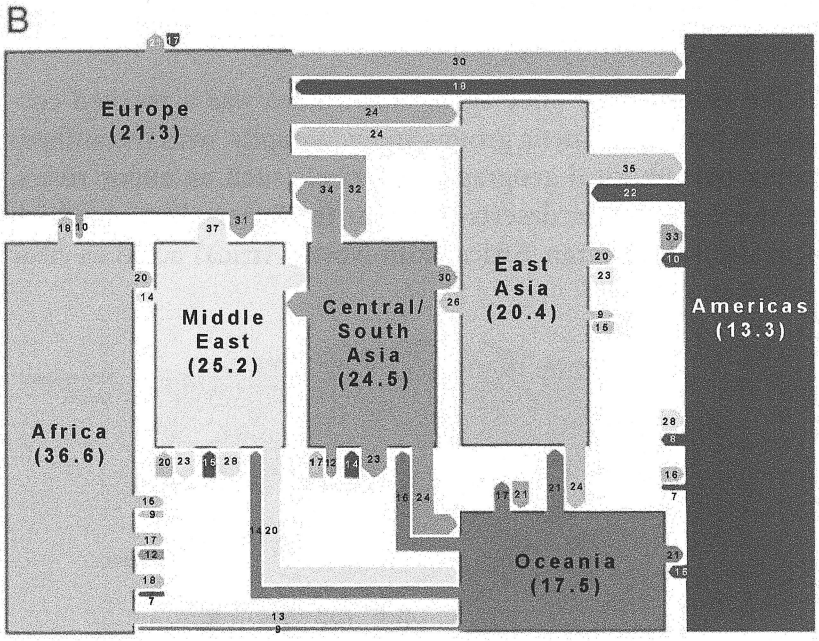


Figure 1B from the paper "Haplotype variation and genotype imputation in African populations," Genetic Epidemiology (2011). Titled "schematic world map of haplotype variation," this paper, based on previous SNP analysis, graphically maps out how SNP analysis divides the earth's population into seven regions, all of which correspond to physical types, or "races."

SNP Analysis Links Geography to Racial Delineation

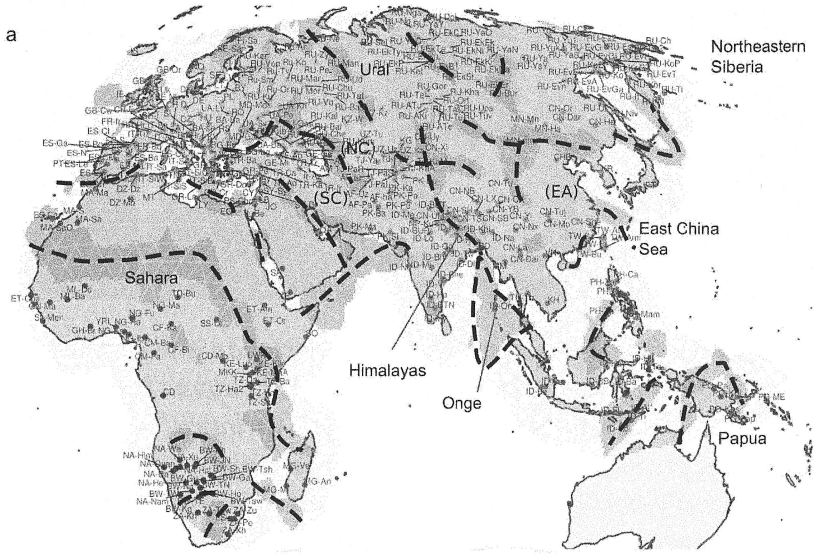
A 2020 study, published in the journal *Molecular Biology and Evolution*,[10] used a new tool known as "Estimated Effective Migration Surfaces" (EEMS)—which was designed to visualize the relationship of genes to geography—to chart "geographic patterns in human genetic diversity."

The paper's authors applied the EEMS system and a principal component analysis (PCA) to a dataset of SNP data combined

10 "Genetic Landscapes Reveal How Human Genetic Diversity Aligns with Geography," Peter, B. M., et al., *Molecular Biology and Evolution*, April 1, 2020; 37(4):943–951.

from 27 different sources comprising thousands of individuals from 419 locations across Eurasia and Africa.

The results were once again clear: there was a marked correlation between genetic groups and geography, with the authors noting that physical geographic barriers (such as major rivers, sea spaces, or major deserts—such as the Sahara Desert, which separates sub-Saharan Africa from North Africa) acted as clear demarcation lines.



From Supplemental Data Figure 2a of the paper "Genetic Landscapes Reveal How Human Genetic Diversity Aligns with Geography," Molecular Biology and Evolution (2020). A map of Africa and Eurasia demonstrates the link between genetic groups and geography.

Yet another study, published in the journal *Science*,[11] used a dataset of 650,000 SNPs from 51 populations in its population overview. Once again, the conclusions were clear: "Individual ancestry and population substructure were detectable with very

11 "Worldwide Human Relationships Inferred from Genome-Wide Patterns of Variation," Jun. Z. Li, et al., *Science,* Feb. 22, 2008, Vol 319, Issue 5866, pp. 1100–1104.

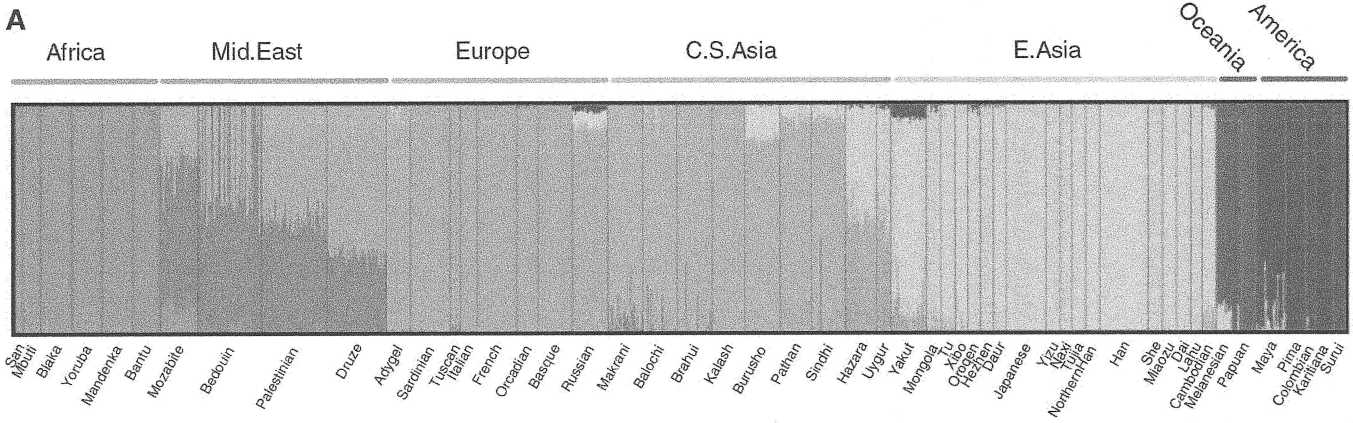


Figure 1 from the paper "Worldwide Human Relationships Inferred from Genome-Wide Patterns of Variation," Science (2008), titled "Individual ancestry and population dendrogram." Using a SNP analysis, the bar clearly divides humans up into seven distinct categories: Africa, the Middle East, Europe, Central/South Asian, East Asia, Oceania, and America. The original caption read: "Each individual is represented by a vertical line partitioned into segments whose lengths correspond to his/her ancestry coefficients in up to seven inferred ancestral groups. Population labels were added only after each individual's ancestry had been estimated; they were used to order the samples in plotting."

high resolution . . . In addition, we observed a pattern of ancestral allele frequency distributions that reflects variation in population dynamics among geographic regions."

These studies—and numerous others—have all served to confirm beyond any question that there are seven distinct racial groups which can be identified upon the basis of DNA studies alone.

Race—as expressed in a group with common physical features—can be determined though a study of shared Y and mtDNA haplogroups, combined with SNP analysis and comparison.

The only remaining genetic question which must be answered is the identification of the genes which are responsible for forming the characteristics which make up the races.

CHAPTER FIVE

RACE AND RACIAL DIFFERENCES CREATED AT THE GENE AND SNP LEVEL

With the biological—that is, the genetic—basis of race and racial categories firmly established, it now remains only to show how specific genes regulate these racial differences.

A short recap of the essential points is helpful before moving on to identifying and listing the actual genes themselves.

1. The sex-determining chromosomes (Y and mtDNA) serve as geographic and racial "family tree" separators;

2. The autosomal DNA chromosomes (and specifically the genes and smaller components, SNPs, exons and nitrons) also clearly display specific geographic and racial affinities, which allow for the definitive identification of seven major races of the world; and

3. Race can therefore be defined as a related group of people whose common physical appearance is determined by genetic factors alone.

List of Defining Physical Racial Characteristics

In order to show exactly how genes create race, it is first required to draw up a list of definitive racial characteristics which are the hallmarks of racial categories. These differences can be divided into physical and psychological attributes. In summary, these are the main identified differences between races:

Skin color. This is possibly the most obvious difference, and the one most widely referred to in any discussion of race. This is so because of the obvious contrast between Africans and Europeans in particular, even though skin color is of varying shades among Europeans themselves, and between other races as well.

Skull shape and structure. One of the deepest racial differences can be seen in the shape of the skull. This affects cranial capacity—that is, brain size, which is also divided up by race, and also includes attributes such nasal width, length, shape, cheek size, upper and lower lip dimensions, and epicanthic folds (of the upper eyelid covering the inner corner of the eye—one of the dominant traits in people of Asiatic decent).

Skeletal structure. This includes the length ratio of arms and legs to torso size, and, consequently, height.

Eye color. The non-European races all tend toward black, or near black eye color. This trait clines toward the European far north, where blue eyes predominate.

Hair color. Another marked difference between the races which is also obvious to the naked eye. The non-European races all tend toward a pitch-black hair color, with perhaps the only notable exception being Australian Aborigines, who display light-colored hair to varying degrees.

Hair texture. Europeans, and those with European descent (such as most Indians from India) tend to have straight, or at best, slightly wavy, hair texture. This is a trait also shared by many Asiatics, Semites (Middle Easterners), and Amerindians. In addition, large numbers of sub-Saharan Africans and South Asians from tropical zones have no facial hair. This trait has replicated out in many Amerindians, Chinese, and Japanese men as well, depending on their ancestry.

Psychological differences: These include the propensity to violence, anti-social behavior, and even defined mental illnesses such as schizophrenia.

Intelligence: There are specific genes which code for intelligence, and this attribute is also clearly defined by race. This is the cause of much invented, politically motivated, but factually incorrect, denial.

All the above traits are controlled by specific genes, and, for obvious reasons, these genes are "expressed" in the different races, giving them their distinctive physical appearance.

In other words, although all humans have 23 pairs of chromosomes (that is, 46 in total), this fact by itself does not determine to which racial group an individual belongs.

Race is primarily determined by physical attributes. These physical attributes are not determined by the overall chromosomal structure, but rather by induvial genes, SNPs, and nitrons at a molecular level of that individual's DNA.

Race Created through Different Geographical Concentration of SNPs and Genes

This then, at SNP and gene level—is where race is created, and where it takes physical form. These are not tiny or irrelevant differences, as the race-denialist lobby repeats *ad infinitum.*

These molecular, biologically based differences create vast changes in racial type, behavior, and appearance.

Once this fact is understood—and, to repeat, it is a biological fact—then the utter falsity of the "all races are 99 percent the same" can be clearly seen.

To argue that all humans are indistinct because they all have the same number of chromosomes is akin to saying that all breeds of dogs "are the same" because they all have 39 pairs of chromosomes (78 in total), or that all cats "are the same" because they all have 19 pairs of chromosomes (38 in total), or that all cows "are the same" because they all have 60 chromosomes.

As everyone knows, a German Shepherd is not the same as a poodle, nor is a whippet the same as a sausage dog, despite those breeds having the same number of chromosomes. It is the ma-

jor differences at molecular level which differentiate those breeds from one another—and this process is the same with humans.

It is another common misconception—motivated perhaps by ignorance, or perhaps though malicious intent—to use individuals of mixed-racial origin to "deny" that unique racial differences exist.

The existence of mixed-racial types, who may exhibit characteristics of more than one racial group (such as blue eyes in a person otherwise displaying a clearly non-European physiognomy), does not "disprove" the existence of different races.

If anything, it shows that races *do* exist, even if only by demonstrating the contrast *between* the parent races within the mixed-race individual.

Ironically, the physical inheritability of racial characteristics is, despite decades of race-denial, one of the most known and understood features of inheritance.

Even people with absolutely no knowledge of genetics, inheritance, or race, will all know that children look like their parents or will show physical features, temperament, and intelligence clearly linked to their direct antecedents.

Because these inherited traits are so well known—and accepted—it seems almost bizarre to have to "prove" them with genetic tests, but such is the current state of race-denial that this is a task which is now required.

CHAPTER SIX

THE GENES WHICH DETERMINE RACIAL DIFFERENCES IN SKIN, EYE, AND HAIR COLOR; HAIR TEXTURE AND SHAPE

Skin, eye, and hair color are another three widespread measures of racial origins, and are often used to divide Europeans, or those with varying degrees of European ancestry. Like all physical traits, skin, hair, and eye color are controlled by the presence of specific genes.

An individual who does not possess the gene for blue eyes, for example, will never have blue-eyed offspring, even if their mate does. The only time a mixed-race individual has blue eyes will be when both parents possess the gene, albeit as a "recessive" gene.

Skin color is generated in the same way. The large mixed-race populations of the world, such as the "African Americans" and "Cape Coloureds" (of South Africa), who both on average have around 25 percent percent European admixture,[1] display a large range of skin tones ranging from very light to very dark—all the result of recessive or dominant genes playing out in these

1 "Blacks" in America have on average 24 percent European DNA, and many have more than that (Katarzyna Bryc, et al., "The Genetic Ancestry of African Americans, Latinos, and European Americans across the United States," *American Journal of Human Genetics,* Jan. 8, 2015; 96(1): 37–53). In South Africa, the "Coloured" population have a similar level of European admixture, between 21 and 28 percent ("Genome-wide analysis of the structure of the South African Coloured Population in the Western Cape," De Wit, E., et al., *Human Genetics* volume 128, pages 145–153 (2010)).

groups. This also happens in other regions with large mixed-race populations, such as those found in Central and South America.

Skin Color: The Genetics of Melanin Production

Many people know that "melanin" is the substance in a human body which produces hair, eye, and skin pigmentation. The basic rule is that the more melanin, the darker the eyes, hair, and skin.

Like all physical attributes, this melanin production is the product of specific genes and SNPs. The actual process is as follows:

A gene called "MC1R" (located at 16q24.3, chromosome 16) provides the instructions for the creation of a protein called the "melanocortin 1 receptor."

This receptor is located on the surface of specialized cells called melanocytes. It is these melanocytes which produce the melanin pigment.

Melanocytes make two forms of melanin, called "eumelanin" and "pheomelanin." Skin and hair color are determined solely by the relative amounts of these two pigments—and these levels are in turn determined by the coding within the MC1R gene.

The melanocortin 1 receptor controls which type of melanin is produced by melanocytes. When the receptor is activated, it triggers a series of chemical reactions inside melanocytes that stimulate these cells to make eumelanin. If the receptor is not activated or is blocked, melanocytes make pheomelanin instead of eumelanin.

Therefore, if an individual's MC1R gene is coded to produce a prodigious amount of melanin, then that individual will have dark hair and skin, and vice versa.

Individuals whose genes produce mostly eumelanin tend to have brown or black hair, while those whose genes produce mostly pheomelanin tend to have red or blond hair, freckles, and light-colored skin.

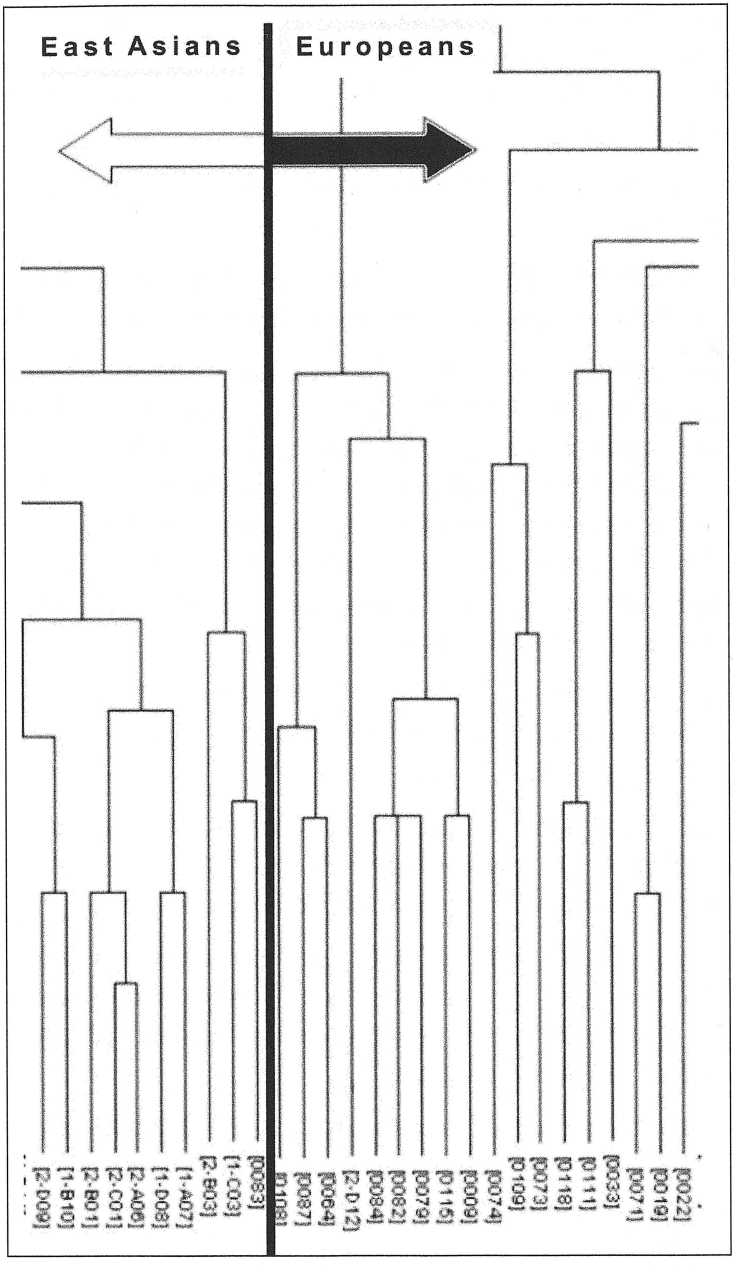


Figure 2 from a 2008 study into racial skin genetics between East Asians and Europeans. The racial groups were separated by a line in the results. Left: Mongoloid subjects; right: Caucasoid subjects.

This provides the key to understanding the origin of racial differences in skin color: once again, like all racial differences, it is a question of differentiation at SNP and gene level which creates racial phenotypes.

Different SNPs Create Different Skin Tones in Europeans and Mongoloids

A 2008 study conducted in Japan set out to identify the specific "SNP alleles associated with racial differences in skin color."[2]

Using samples from "122 Caucasians in Toledo, Ohio and 100 Mongoloids in Japan," the scientists looked for 20 SNPs in 7 candidate genes and a number of previously identified proteins, which included the "Agouti signaling protein" (ASIP), the "tyrosinase-related protein 1 "(TYRP1), the tyrosinase (TYR) gene, the MC1R gene, the "oculocutaneous albinism II" (OCA2) gene, the "microphthalmia-associated transcription factor" (MITF), and the "myosin VA" (MYO5A) gene.

The results of their study were stark: the racial groups were definitively split along genetic lines, showing a clear differentiation according to racial origin.

A 2007 study, also conducted in Japan, further investigated racial differences in two coding SNPs, c.G1129A (R163Q) at the MC1R gene and c.A1962G (H615R) at the OCA2 gene.[3]

The study found that the "Q163 and R615 alleles prevailed almost exclusively in East and Southeast Asian populations" and that the "frequency of the Q163 allele was higher in Northeast Asians than in Southeast Asians."[4]

Furthermore, the study continued, the L374F allele—a mutation—which is found in the transporter protein of the SLC45A2

2 "Interactions between SNP Alleles at Multiple Loci Contribute to Skin Color Differences between Caucasoid and Mongoloid Subjects," Anno, S., et al., *International Journal of Biological Sciences,* 2008; 4(2): 81–86.
3 "Distribution of Two Asian-Related Coding SNPs in the MC1R and OCA2 Genes," Yuasa, I., et al., *Biochemical Genetics* 45:535–542, 2007.
4 *Ibid.*

gene (located at 5p13.2, chromosome 5), and A111T mutation of that same gene, is found only in Europeans.[5] Unsurprisingly, the study also found that the Q163 allele is "very common in New World populations [that is, in Amerinds] which have an affinity with East Siberian and Northeast Asian populations."[6]

Furthermore, the R615 allele at the OCA2 locus was rare or not found in Indo-European and African populations, whereas it was very common in Asian populations. Other Asian-specific mutations include the alleles rs12438490, rs12440330, rs12442916, rs1545397, rs12439410, and rs12440423, all located in intronic regions of the OCA2 gene.

"These mutations are observed at extremely high frequencies only in the Chinese and Japanese," the study continued. [7]

In Europeans, the KITLG gene (located at 12q21.32, chromosome 12) has also been identified as contributing toward their skin color.[8]

Different SNPs Create Different Skin Tones in Sub-Saharan Africans and San ("Bushmen")

A 2017 study on the origin of skin pigmentation in Africa observed the lowest melanin levels in southern African "San hunter-gatherers" (previously called "Bushmen") and highest levels in East African Nilo-Saharan pastoralists.[9]

This variation was studied under a genome-wide association study of 1570 Africans which revealed that variant alleles "significantly associated with skin pigmentation" clustered in four genomic regions on that continent.

These genes are:

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5 *Ibid.*
6 *Ibid.*
7 *Ibid.*
8 "The Timing of Pigmentation Lightening in Europeans," Beleza, S., *Molecular Biology and Evolution,* Volume 30, Issue 1, January 2013, Pages 24–35.
9 "Loci associated with skin pigmentation identified in African populations," Crawford, N. G. et. al., *Science,* Oct. 12, 2017, Vol 358, Issue 6365.

- The gene MFSD12 (located at 19p13.3, chromosome 19) which is linked to very dark skin;

- The gene SLC24A5 gene, which is linked to lighter skin and was introduced to East Africa from the Middle East in an unknown migration around five thousand years ago;

- The DDB1 gene (located at 11q12.2, chromosome 11), which is linked to repair from UV damage—a rarity among dark-skinned peoples, and

- A mutation in the OCA2 and HERC2 genes observed only in the San people.

The study also revealed that the dark-skinned South Asian and Australo-Melanesian populations (in Borneo, for example) also have the identical MFSD12 gene—which means that at some stage they must have had a common ancestor.

From the above it is clear that specific genes create skin color, and that these genes are unique to certain racial groups, even when, as is the case with many Asians and Europeans, they share a light skin color.

The Genetics of Hair Color: The Genes which Cause Black, Brown, Blond and Red Hair

Just as the case with skin color, hair color is also determined by the MC1R gene in particular.

If an individual's gene produces mostly eumelanin, his or her hair color will be dark (black or brown, depending upon the amount of eumelanin produced). If the gene produces mostly pheomelanin, the hair color will be light. If an abundance of pheomelanin is produced, the hair color will be orange or red.

Jet-black hair, as is common outside Europe, is produced by tightly packed eumelanin, while red hair is produced by tightly packed pheomelanin. Blonde hair has both types of melanin, but in very small amounts and loosely packed.

The graying of hair occurs when a decline in pigment cells occurs, and less and less melanin is stored. Gray hair is a sign of little pigment, while white hair has no pigment.

Like all genes, these melanin producing cells are passed down from parent to child. The variability of hair color in a family depends directly on their direct ancestors, and this is why European children in the same family can have different color hair. Outside of Europe, this does not happen, unless the individuals in question have some European ancestry themselves.

Other genes which have been shown to control levels of eumelanin and pheomelanin production include ASIP (located at 20q11.22, chromosome 20), DTNBP1 (located at 6p22.3, chromosome 6), GPR143 (located at Xp22.2, X chromosome), HPS3 (located at 3q24, chromosome 3), KITLG (located at 12q21.32, chromosome 12), MLPH (located at 2q37.3 , chromosome 2), MYO5A (located at 15q21.2, chromosome 15), MYO7A (located at 11q13.5, chromosome 11), OCA2 (located at 15q12-q13.1, chromosome 15), SLC45A2 (located at 5p13.2, chromosome 5), SLC24A5 (located at 15q21.1, chromosome 15), TYRP1 (located at 9p23, chromosome 9), TYR (located at 11q14.3, chromosome 11), ERCC6 (located at 10q11.23, chromosome 6), GNAS (located at 20q13.32, chromosome 20), HERC2 (located at 15q13.1, chromosome 15), IRF4 (located at 6p25.3, chromosome 6), OBSCN (located at 1q42.13, chromosome 1), SLC24A4 (located at 14q32.12, chromosome 14), TPCN2 (located at 11q13.3, chromosome 11), and MITF (located at 3p13, chromosome 3).[10]

The study went on to link specific SNPs and alleles with hair colors:

SNP rs16891982, gene SLC45A2, black hair.

SNP rs28777, gene SLC45A2, black hair.

SNP rs26722, gene SLC45A2, black hair.

SNP rs12203592, gene IRF4, black hair.

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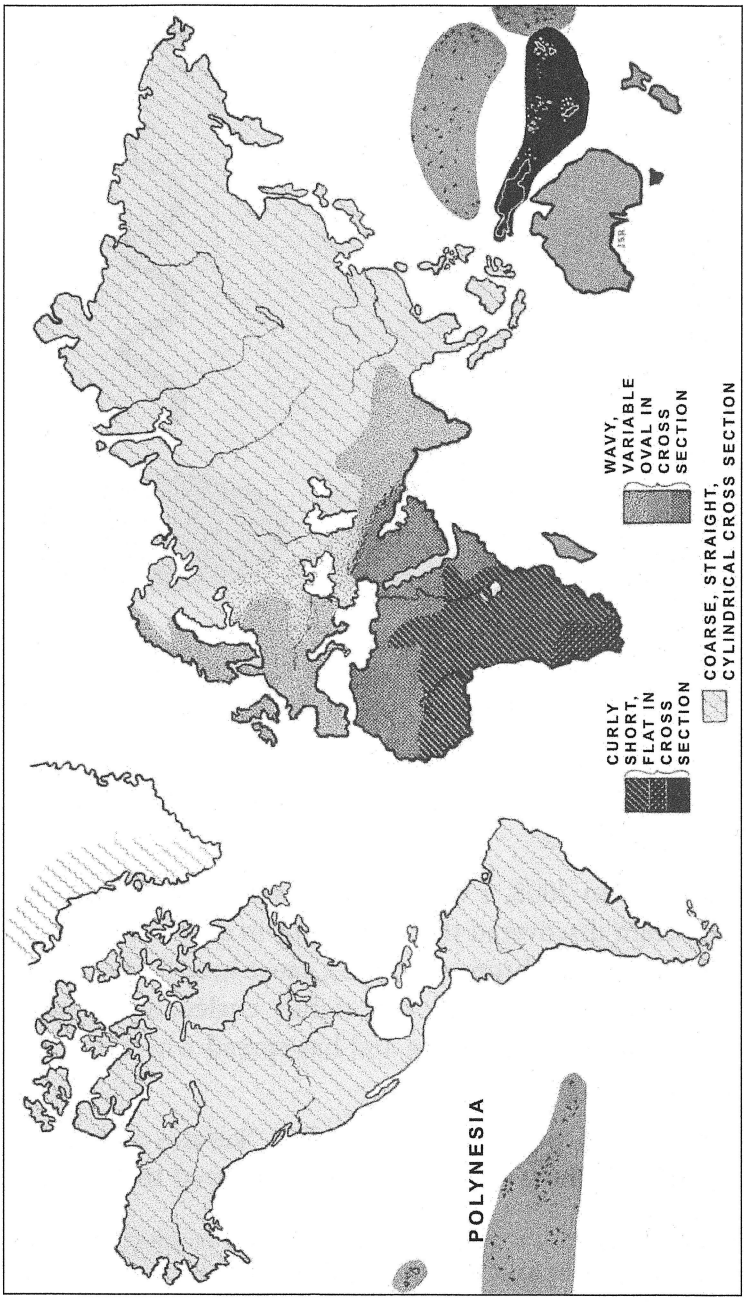
10 "Model-based prediction of human hair color using DNA variants," Branicki, W., et al., *Human Genetics,* 2011; 129(4): 443–454.

SNP rs9378805, gene IRF4, black hair.
SNP rs4959270, gene EXOC2, black hair.
SNP rs12913832, gene HERC2, black hair.
SNP rs1393350, gene TYR, brown hair.
SNP rs4778138, gene OCA2, brown hair.
SNP rs4904868, gene SLC24A4, blond hair.
SNP rs1805005, gene MC1R, blond hair.
SNP rs2402130, gene SLC24A4, dark-blond hair.
SNP rs1800407, gene OCA2, red hair.
SNP rs916977, gene HERC2, red hair.
SNP rs8039195, gene HERC2, red hair.
Allele MC1R_R, gene MC1R, red hair.
Allele MC1R_r, gene MC1R, red hair.
SNP N29insA16, gene MC1R, red hair.
SNP rs2228479, gene MC1R, red hair.
SNP rs11547464, gene MC1R, red hair.
SNP rs1805007, gene MC1R, red hair.
SNP rs1805008, gene MC1R, red hair.
SNP rs1805009, gene MC1R, red hair.
SNP rs2378249, gene ASIP, red hair.
SNP rs1015362, gene ASIP, red hair.
SNP rs1015362, gene ASIP, blood-red hair.

Europeans are highly differentiated from all other races in that they have a far wider hair color range—and therefore different hair color genetics. The same goes for eye color.

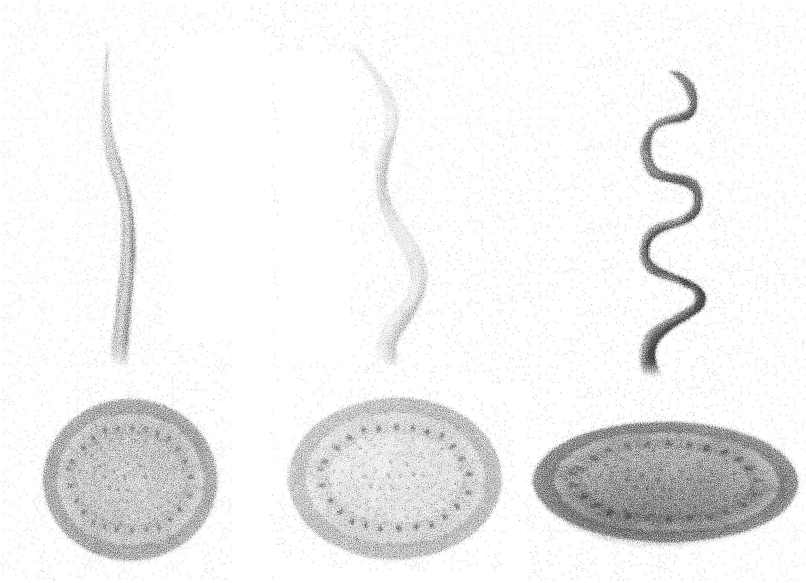
The Genetics of Hair Texture: The Genes which Cause "Round" European Hair, "Oval" Asian Hair, and "Flat" African Hair

Hair texture, like skin color and eye color, is also clearly racially graded. A "hair map" of the world illustrates this reality:



A "hair map" of the world.

It is a little fact that even the shape of individual hairs—flat, oval, or round, depends upon race.



Hair differs physically by race: from left to right: European hair, Asian hair and African hair.

Genes cause these physical differences, and numerous studies have demonstrated exactly which genes are at work in creating these racial differences.

African hair: In 2008, a Columbia University Medical Center research team discovered that mutations in a gene, known as P2RY5 (13q14.2–14.3, chromosome 13), cause hereditary "woolly hair,"[11] or hair that is coarse, dry, tightly curled, and sparse. This gene is common to Africans and has spread wherever they have left a genetic legacy.

All hair contains proteins known called "keratin-associated protein" (KAP) clusters, but a 2014 study[12] showed that certain

11 "Disruption of P2RY5, an orphan G protein-coupled receptor, underlies autosomal recessive woolly hair," Shimomura, Y., *Nature Genetics,* volume 40, pages 335–339 (2008).

12 "Geographic Distribution and Adaptive Significance of Genomic Structur-

variations in the KAP cluster genes are present in African populations but virtually absent elsewhere—and that they contribute to the unique appearance and texture of African hair.

Asian hair: Scientific studies have shown that variations in two genes, EDAR (location 2q11-q13, chromosome 2) and FGFR2 (10q26.13, chromosome 10) are the drivers of hair thickness, straightness, and texture in Asians.[13]

European hair: Research has revealed that a polymorphism in the gene, TCHH (location 1q21.3, chromosome 1), codes for hair texture in people of northern European ancestry.[14] The cylindrical shape of this European hair is created by the way TCHH binds with other proteins and to keratin molecules.

The Genes Which Cause Blue, Brown, and Black Eyes—and the Ranges In Between

There are, obviously, a wide range of eye colors, ranging from blue to gray to green to hazel to brown to dark brown to black—and almost any variation between these absolute colors. These variations are always however the result of mixing at some stage in an individual's ancestry.

The genetics behind eye color have now long been established. While at least 16 genes play a role in determining eye color, two of these genes—called OCA2 and HERC2, located at 15q12-q13.1 and 15q13 (Chromosome 15) respectively, are the most important. A 2006 study, for example, showed that a "quantitative-trait locus linked to the OCA2 region of 15q accounts for 74 percent of variation in human eye color . . ."[15]

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al Variants: An Anthropological Genetics Perspective," Eaaswarkhanth, M., Human Biology, Vol. 86, No. 4 (Fall 2014), pp. 260–275.

13 "A scan for genetic determinants of human hair morphology: EDAR is associated with Asian hair thickness," Fujimoto, A., *Human Molecular Genetics,* Mar. 15, 2008; 17(6):835–43.

14 "Common variants in the trichohyalin gene are associated with straight hair in Europeans," Medland, S.E., et al., *American Journal of Human Genetics,* Nov. 2009; 85(5):750–5.

15 "A three-single-nucleotide polymorphism haplotype in intron 1 of OCA2 explains most human eye-color variation," Duffy, D., et al., *American Journal*

The study went on to analyze a dataset made up of 58 "exonic [exon] single-nucleotide polymorphisms (SNPs) and tagging SNPs" from thousands of subjects. They found that the "highest association for blue/non-blue eye color was found with three OCA2 SNPs: rs7495174 T/C, rs6497268 G/T, and rs11855019 T/C . . . in intron 1."[16]

"These three SNPs are in one major haplotype block, with TGT[17] representing 78.4 percent of alleles.

"The TGT/TGT diplotype[18] found in 62.2 percent of samples was the major genotype seen to modify eye color, with a frequency of 0.905 in blue or green compared with only 0.095 in brown eye color."[19]

The study added that this blue eye genotype "was also at highest frequency in subjects with light brown hair and was more frequent in fair and medium skin types, consistent with the TGT haplotype acting as a recessive modifier of lighter pigmentary phenotypes."[20]

A 2007 study carried out in the Netherlands confirmed this finding, reporting that the SNPs in the HERC2 gene (located at 15q13), and "to a lesser extent," in the neighboring OCA2 gene (located at 15q12-q13.1) were independently associated to iris color variation.[21] In addition, the HERC2 rs916977 SNP "showed a clinal allele distribution across 23 European populations, which was significantly correlated to iris color variation."[22]

Another study showed that the SNP rs916977 (chromosome 15) is found in 97 percent of all Europeans with blue eyes, and variations in SNPs rs1129038 and rs12913832 are relatively com-

of Human Genetics, Feb. 2007; 80(2):241–52.

16 *Ibid.*

17 Transglycosylases, or TGT, is an enzyme that serves to transform molecules containing sugar into other molecules.

18 A diplotype is a combination of two haplotypes.

19 *Ibid.*

20 *Ibid.*

21 *Ibid,* page 411.

22 *Ibid.*

mon in Caucasians "though rare among other ethnic groups."[23]

Yet another study revealed that "a single SNP in intron 86 of HERC2, rs12913832, predicted eye color" as blue or brown.[24]

In 2009, it was revealed that a "missense"[25] SNP (rs1800407) is associated with green/hazel eyes.[26]

A 2012 study investigated the "eye-color genes" on an international level.[27] This study found that "the blue-eye associated alleles at all three haplotypes were found at high frequencies in Europe; however, one is restricted to Europe and surrounding regions, while the other two are found at moderate to high frequencies throughout the world."[28]

The study also found that the "allele of rs1800414 is essentially limited to East Asia where it is found at high frequencies"[29]—in other words, this is the source of Asian eye color.

All this was confirmed in a 2010 study[30] which found that while there are 16 different genes responsible for eye color in humans, the two most important ones were those located on chromosome 15. As the study revealed, the "single-nucleotide poly-

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23 "Blue eye color in humans may be caused by a perfectly associated founder mutation in a regulatory element located within the HERC2 gene inhibiting OCA2 expression," Eiberg, H., et al., *Human Genetics*, 2008 March; 123(2):177–87.

24 "A single SNP in an evolutionary conserved region within intron 86 of the HERC2 gene determines human blue-brown eye color," Sturm, R. A., *American Journal of Human Genetics*, Feb. 2008; 82(2):424–31.

25 A genetic alteration in which a single base pair substitution alters the genetic code in a way that produces an amino acid that is different from the usual amino acid at that position.

26 "Interactions between HERC2, OCA2 and MC1R may influence human pigmentation phenotype," Branicki, W., at al., *Annals of Human Genetics*, Mar. 2009; 73(2):160–70.

27 "A global view of the OCA2-HERC2 region and pigmentation," Michael P. Donnelly, M. P., et al., *Human Genetics,* 2012; 131(5): 683–696.

28 *Ibid.*

29 *Ibid.*

30 "Genotype-phenotype associations and human eye color," White, D., et al., *Review Articles, Journal of Human Genetics – Nature,* Jan. 2011; 56(1):5–7.

morphisms in either of these two genes have a large role in the eye color of an individual."[31]

The mechanics of the process are complex, but they are in essence the chemical process by which melanin is created. The most important genes which control this process are known by their technical names of "'Homologous to the E6-AP Carboxyl Terminus" or "HECT" region (or "domain"), and the "Regulator of chromosome condensation 1," or "RCC1" domain.

They contain the protein called "HERC2" (referred to above, and located at 15q13, chromosome 15), which is encoded by 93 exons.

An intron in HERC2 provides what is called the "promoter region" for the gene named OCA2. The OCA2 gene provides instructions for making a protein called the "P protein" which is found in specialized cells called melanocytes.

These melanocytes produce the pigment called melanin—which is the substance that gives skin, hair, and eyes their color.

In other words, the level of melanin in an individual is coded at SNP level within their genes—and this directly affects their skin tone, eye, and hair color.

Finally, a 2012 study found that the HERC2 (OCA2) allele is absent from African and East Asian populations and appears at high frequency only in Western and Northern Europe. The absence of this allele from African and East Asian populations explains their dominant eye color.

Eye Color is Dependent Upon the Presence of Specific SNPs

The evidence is clear: the eye color of an individual depends exclusively upon the existence or otherwise of specific SNPs within specific genes—and these genes are inherited directly from that individual's parents. When these inherited genes are shared by enough people, they create a "family" or similar looking phenotype—what we call a race.

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31 *Ibid.*

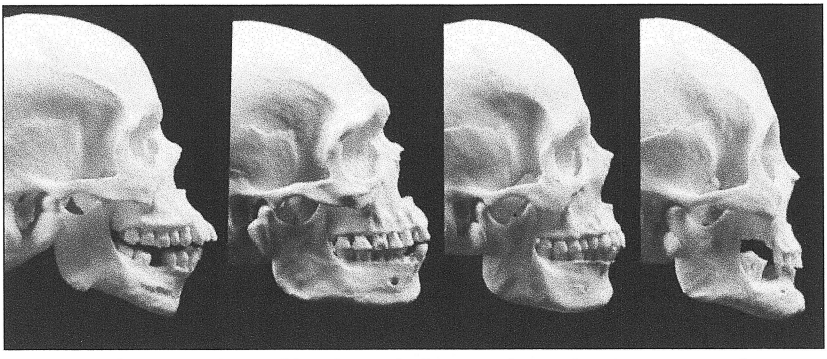
CHAPTER SEVEN

THE GENES WHICH DETERMINE RACIAL DIFFERENCES IN SKULL STRUCTURE AND FACIAL FEATURES

Although skull shape is one of the easiest racial identifiers available to forensic scientists, this racial reality is largely unknown to the general public. If it is ever mentioned in public, then it is generally portrayed as some type of ancient, wicked, or discredited pseudo-science.

Differences in Skull Shapes are used by Forensic Anthropologists to Determine Race

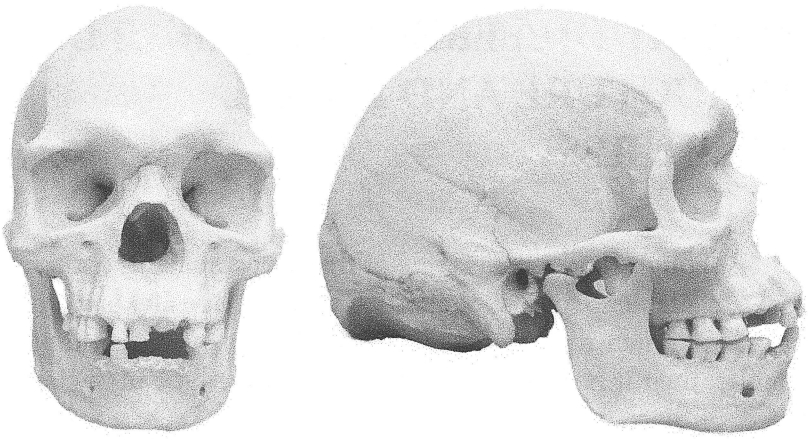
Nothing, of course, could be further from the truth, and all police forensics, anatomists, and medical professionals are aware of important differences in skull shape. These obvious differences are actively taught in medical schools and in the forensic scienc-



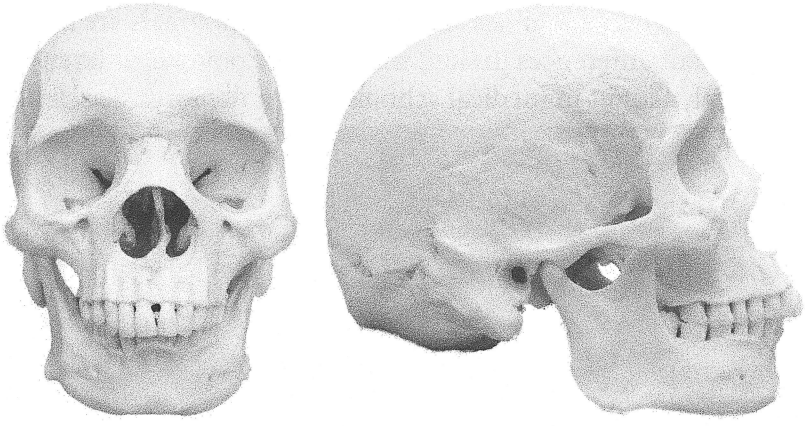
Racial types in profile: from left to right, African, Australian Aborigine, European, and Amerindian.

es. Like all physical traits, skull shape is determined by the genetics of an individual.

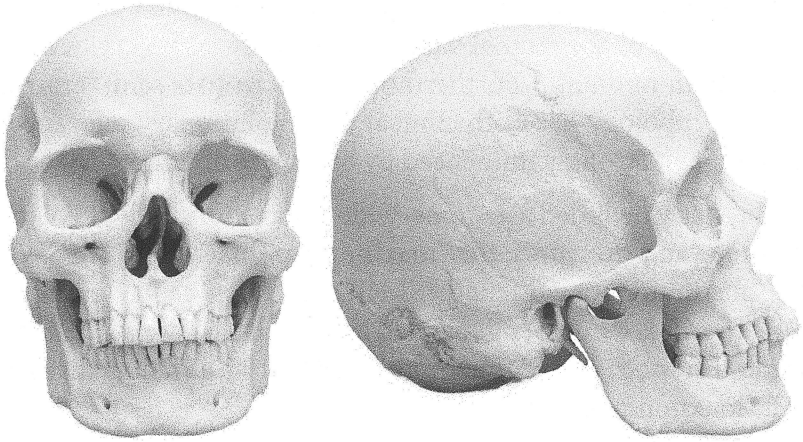
These genes are inherited from that individual's parents, and when these genes are shared among enough people, they form a "group"—or a "race."



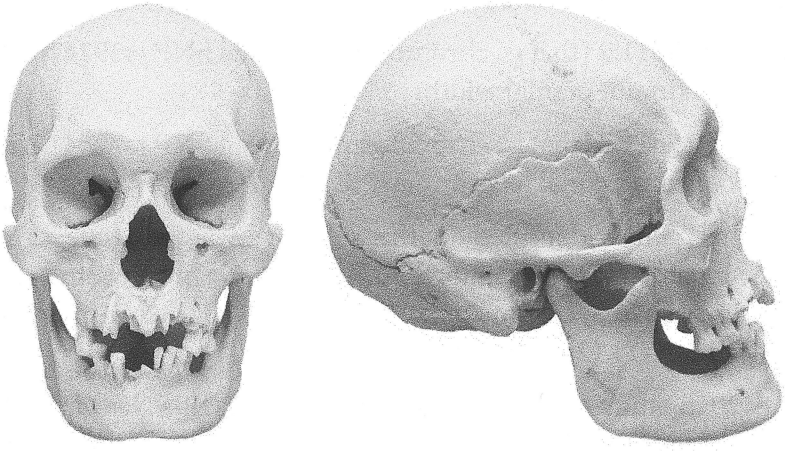
Typical Australian Aborigine Skull



Typical Sub-Saharan African Skull



Typical European Skull (Northwestern Europe)



Typical Polynesian Skull

The Genes which are Responsible for Different Racial Skull Shapes

There are also many scientific studies which have conclusively demonstrated the link between genetics and skull shape and structure. Below follows an overview of the most prominent such studies, all of which come to the same conclusion: that specific genes are responsible for cranial formations.

In 2017, a team of international scientists carried out a study of the "shape of the cranial vault"[1] which they said, "varies considerably in humans."[2] Furthermore, the scientists said, "cranial vault morphology has both clinical and evolutionary relevance" because it is "strongly influenced by brain size and shape."[3]

The study included a genome-wide association study (GWAS) on three vault measures: the "maximum cranial width" (MCW), the "maximum cranial length" (MCL), and the "cephalic index" (CI). The CI is a number generated by calculating the ratio of the maximum width of the head, multiplied by 100, and then divided by its maximum length.

The sample was taken from "4419 healthy individuals of European ancestry" and the results revealed the specific genes responsible for creating the skull shapes:

Gene 15p11.2 (that is, chromosome 15, lead SNP rs29247670-8) for maximum cranial width.

Gene 17q11.2 (that is, chromosome 17, lead SNP rs72841279) for maximum cranial length.

Other genes identified in the study which influence skull shape included the ones named NLK (17q11.2, chromosome 17), MEF2A (15q26.3, chromosome 15), SOX9 (17q24, chromosome 17), and SOX11 (2p25.2, chromosome 2).

A 2015 study of African Bantus from Tanzania demonstrated "pairwise genetic correlations of 38 measures of facial shape and size," and "absolute genetic correlation between most traits, indicating large overlap in underlying genetic loci."[4] The study analyzed the genotypes of 15 million common SNPs, both in direct family units and in the broader community of their chosen subjects. Their conclusion was that "facial shape and size pheno-

1 "Mapping genetic variants for cranial vault shape in humans," Roosenboom, J., et al., *Plos One,* Apstril 26, 2018.
2 *Ibid.*
3 *Ibid.*
4 "Human Facial Shape and Size Heritability and Genetic Correlations," Cole, J., et al., *Genetics,* February 1, 2017, vol. 205 no. 2 967–978.

types are highly heritable, and additionally are highly genetically correlated, and that a large fraction of the genetic component of facial differences can be explained by common variation genome-wide."

The study also found that "the same alleles that cause an individual to have a broad face also cause that individual to have a short face, and vice versa."

The Genes Which are Responsible for Phenotype in Facial Structure

An international study conducted in 2012 started off its work with the announcement that "inter-individual variation in facial shape is one of the most noticeable phenotypes in humans, and it is clearly under genetic regulation study. . ."[5]

The scientists undertaking the research "identified five independent genetic *loci* associated with different facial phenotypes, suggesting the involvement of five candidate genes—PRDM16, PAX3, TP63, C5orf50, and COL17A1—in the determination of the human face." PRDM16 is located at 1p36.32 (Chromosome 1), PAX3 at 2q36.1 (Chromosome 2), TP63 at 3q28 (chromosome 3), C5orf50 at 12q21.32 (chromosome 12), and COL17A1 at 10q25.1 (chromosome 10).

A 2016 study conducted in Tanzania looked for "genetic association of facial shape and size phenotypes."[6] That region was chosen because it is "both genetically and environmentally relatively homogeneous."[7] The study revealed that SNPs within the genes SCHIP1 and PDE8A were associated with measures of facial size in both the genome-wide associated study of facial morphology, and "ten additional loci demonstrated suggestive association with various measures of facial shape."

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5 "A Genome-Wide Association Study Identifies Five Loci Influencing Facial Morphology in Europeans," Liu, F., *Plos Genetics,* September 13, 2012.
6 "Genomewide Association Study of African Children Identifies Association of SCHIP1 and PDE8A with Facial Size and Shape," Cole, J. B., et al., *Plos Genetics,* August 25, 2016.
7 *Ibid.*

Significantly, for an understanding of racial differences, the study went on to state that its "findings, which differ from those in previous studies of European-derived whites, augment understanding of the genetic basis of normal facial development and provide insights relevant to both human disease and forensics."[8]

A 2016 study carried out on European-Americans[9] which included an analysis of one million genotyped SNPs, revealed "genome-wide significant associations for cranial base width at 14q21.1 [that is, on chromosome 14]. and 20q12 [chromosome 20], intercanthal width at 1p13.3 [chromosome 1] and Xq13.2 [x chromosome for females], nasal width at 20p11.22 [chromosome 20], nasal ala length [the bottom side part of the nose] at 14q11.2 [chromosome 14], and upper facial depth at 11q22.1 [chromosome 11]."[10]

Furthermore, the study continued, "several genes in the associated regions are known to play roles in craniofacial development or in syndromes affecting the face: MAFB, PAX9, MIPOL1, ALX3, HDAC8, and PAX1. We also tested genotype-phenotype associations reported in two previous genome-wide studies and found evidence of replication for nasal ala length and SNPs in CACNA2D3 and PRDM16."[11]

A 2018 study revealed that at least 24 SNPs were associated with facial structure, and that a "global map of derived polygenic face scores assembled facial features in major continental groups consistent with anthropological knowledge."[12]

(In other words, the genetic differences matched the physical differentiation of humans into geographically separate races.)

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8 *Ibid.*

9 "Genome-Wide Association Study Reveals Multiple Loci Influencing Normal Human Facial Morphology," Shaffer, J. R., et al., *Plos Genetics,* August 25, 2016.

10 *Ibid.*

11 *Ibid.*

12 "Novel genetic loci affecting facial shape variation in humans," Xiong, Z., et al., *eLife,* Nov. 26, 2019; 8:e49898.

The Genes Which Determine Face Shape, Nose Shape, Eye Position, and All Other Facial Features

It is of course not just the skull shape that is determined by individual genes. The features on the face—width, nose shape, eye positions, and all other physical characteristics are genetically determined.

The 50 or so genes responsible for these variations were identified in a 2013 study of "human facial diversity"[13] in population samples from individuals of mixed West African and European ancestry from three locations (United States, Brazil, and Cape Verde).

This study found that "the independent effects of particular alleles on facial features can be uncovered,"[14] and that "results on a set of 20 genes showing significant effects on facial features provide support for this approach as a novel means to identify genes affecting normal-range facial features and for approximating the appearance of a face from genetic markers."[15]

The Genes Which Cause Epicanthic Folds in Certain Asian Racial Subgroups

Finally, the epicanthic fold, which "represents one of the most distinguishing features of East Asian faces,"[16] has been tracked via a genome-wide association study to two specific SNPs (rs12570134 and rs1415425). A study showed these two SNPs as having "genome-wide significant association with the double eyelid phenotype" among the Japanese people. Among Chinese Han people—the dominant sub-racial group in China—this trait was linked mainly to the rs1415425 SNP.

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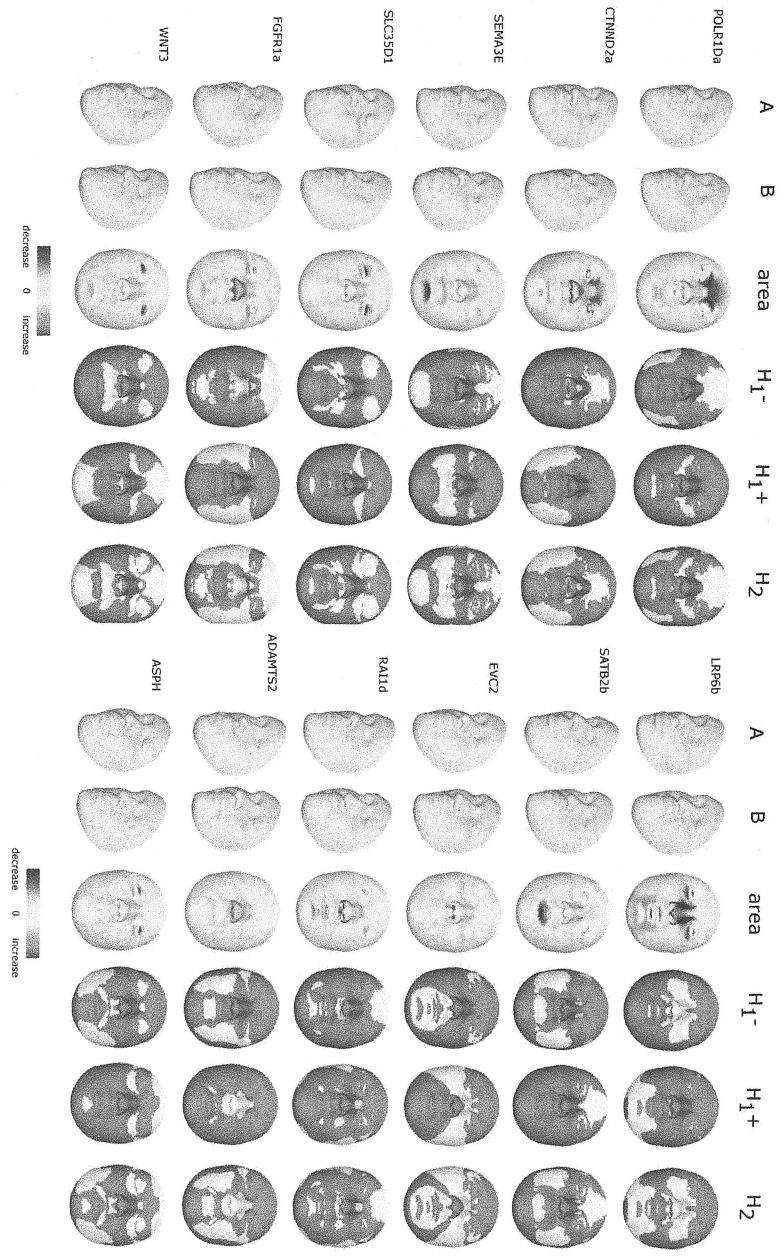
13 "Modeling 3D Facial Shape from DNA," Claes, P., *Plos Genetics,* March 20, 2014.

14 *Ibid.*

15 *Ibid.*

16 "DNA-based eyelid trait prediction in Chinese Han population," Wang, Q., et al., *International Journal of Legal Medicine,* 2021 Sep; 135(5):1743–1752.



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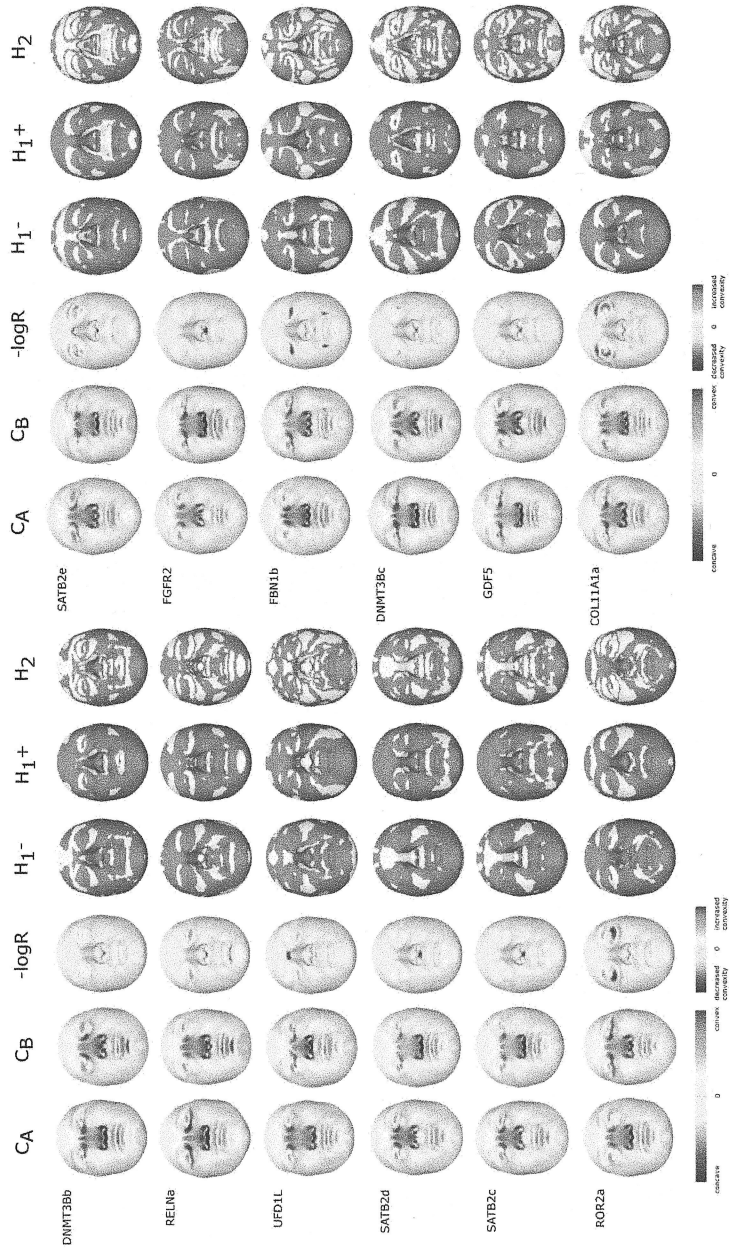
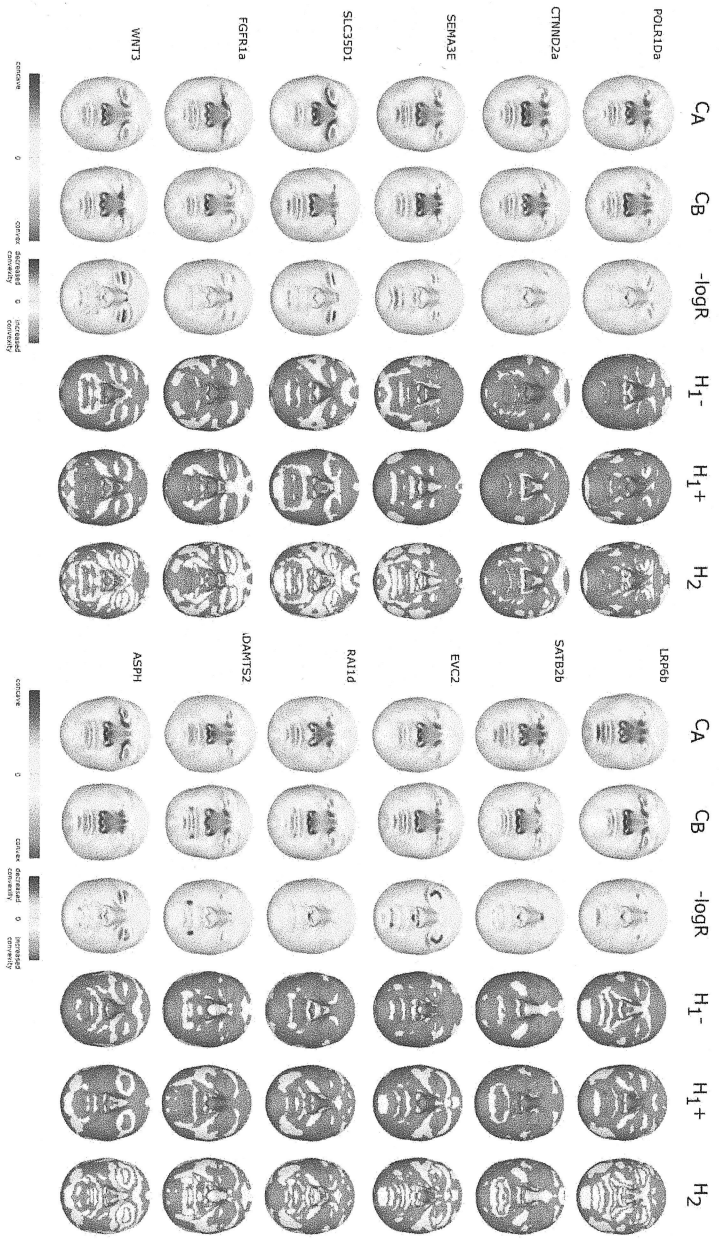


illustrations) upon various parts of the face. The application, or "expression," of these genes upon an individual shapes physical appearance, and thus indicates the race of that individual.



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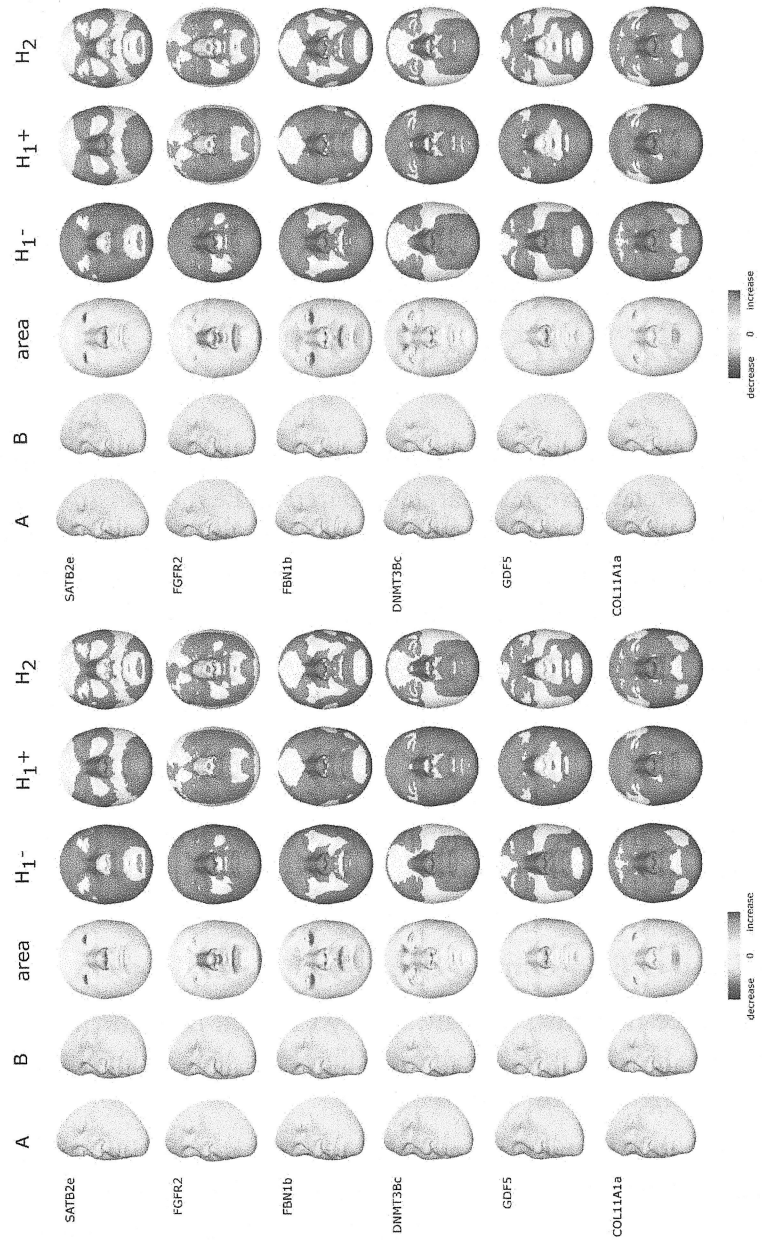

illustrations) upon various parts of the face. The application, or "expression," of these genes upon an individual shapes physical appearance, and thus indicates the race of that individual.

CHAPTER EIGHT

THE GENES WHICH DETERMINE RACIAL DIFFERENCES IN SKELETAL STRUCTURE

Racial differences in height are most striking when northwestern Europeans are compared to East Asians or many South American Amerindians—and indeed, are the subject of common knowledge to the point where these differences are often the topic of impolite jokes.

However, these obvious differentiations are in fact the tip of the genetic iceberg when it comes to skeletal differences between the races.

As discussed earlier, the gene variants in each race produce drastically different skull shapes, and there is no difference when it comes to the rest of the body. Once again, it is genetics, and genetics alone, which determines this reality.

The Limit of Environment Upon Skeletal Development

The race-denialists will always try to claim that nutrition (or rather the lack of) is to blame for differences such as height, arm-to-body length ratios, and so on.

While it is certainly true that insufficient nutrition can and will cause growth problems in any individual, proper eating habits by themselves will never change the pre-determined genetic potential of that individual.

In other words, if an individual's genes are preprogrammed for a certain height, no amount of nutrition (or lack thereof) is

going to change that potential. That person is never going to be taller than his or her genes allow, no matter what the environment.

In addition to this, the other problem that "nurture over nature" race-denialists face is the fact that, on average, for all races, males are taller than females. This is once again predetermined by genetics, and has nothing to do with nutrition, a fact which is obvious from males and females of the same group who share the same environment.

Average Height by Race

A height comparison chart between the different geographical regions highlights this fact:

Average Height by World Region, 2020

	Male	**Female**			**Male**	**Female**
Africa	**Height**	**Height**		**Asia**	**Height**	**Height**
Northern Africa	1.73 m	1.61 m		Western Asia	1.72 m	1.59 m
Eastern Africa	1.67 m	1.57 m		East Asia	1.74 m	1.62 m
Western Africa	1.70 m	1.59 m		South Asia	1.66 m	1.55 m
Southern Africa	1.69 m	1.59 m		Central Asia	1.71 m	1.60 m
Central Africa	1.69 m	1.58 m		Southeast Asia	1.67 m	1.55 m
	Male	**Female**			**Male**	**Female**
America	**Height**	**Height**		**Europe**	**Height**	**Height**
Caribbean	1.74 m	1.61 m		Western Europe	1.80 m	1.66 m
North America (non-European)	1.77 m	1.63 m		Southern Europe	1.76 m	1.63 m

South America	1.73 m	1.60 m		Northern Europe	1.79 m	1.65 m
Central America	1.69 m	1.57 m		Eastern Europe	1.78 m	1.65 m
	Male	**Female**			**Male**	**Female**
Oceania	**Height**	**Height**		**Australia**	**Height**	**Height**
Polynesia	1.76 m	1.65 m		Australia/ New Zealand	1.78 m	1.65 m
Melanesia	1.64 m	1.57 m				
Micronesia	1.69 m	1.59 m				

Source: NCD Risk Factor Collaboration (NCD-RisC).

The tallest people in the world are the Dinkas of Sudan who have an average height of 181.6 cm—and who keep at this height because they remain relatively genetically homogenous—while the Pygmies of Congo, who have an average height of less than 150 cm, are the shortest. Among the European population, Sardinians are the shortest, and the northern Dutch are the tallest.[1]

The Genes Which Determine Height

An international study undertaken in 2010[2] found that there at least hundreds of loci containing height determining genes. Some of the more important ones (but not all) include the following:

ACAN (location 15q26.1, chr. 15, SNPs rs2280470, rs16942341)
SOCS2 (location 12q, chr. 12, SNPs rs10859563, rs11107116)
PML (location 15q24.1, chr. 15, SNPs rs750460, rs5742915)

1 "Worldwide variation in human growth and the World Health Organization growth standards: A systematic review," Natale V., et al., *British Medical Journal*, 2014; 4:e003735.

2 "Hundreds of variants clustered in genomic loci and biological pathways affect human height," Allen, H. L., et al., *Nature*. Oct. 14, 2010; 467(7317): 832–838.

HMGA1 (location 6p21.31, chr. 6, SNPs rs6938239, rs2780226)
GHSR (location 3q26.31, chr. 3, SNPs rs7652177, rs572169)
PPIF (location 10q22.3, chr. 10, SNPs rs7916441, rs2145998)
NPR3 (location 5p13.3, chr. 5, rs3792752, rs1173727)
SDR16C5 (location 8q12.1, chr. 8, SNPs rs10958476, rs7460090)
HHIP (location 4q31.21, chr. 4, SNPs rs2353398, rs7689420)
LCORL (location 4p15.31, chr. 4, SNPs rs2724475, rs6449353)
GH region (location 17q, chr. 17, SNPs rs2070776, rs2665838)
NOG (location 17q22, chr. 17, SNPs rs1401796, rs227724)
FBXW11 (location 5q35.1, chr. 5, SNPs rs6892884, rs12153391)
EFEMP1 (location 2p16.1, chr. 2, rs1367226, rs3791675)
DNM3 (location 1q24.3, chr. 1, rs2421992, rs17346452)
GPR126 (location 6q24.2, chr. 6, SNPs rs225694, rs7763064)
IHH (location 2q35, chr. 2, SNPs rs10187066, rs12470505)
MICA (location 6p21.33, chr. 6, SNPs rs879882, rs2256183

Note: this is a partial list. There are many more such loci, and the large number which affect height mean that the trait is highly heritable—which in turn means that there is a great deal of commonality among the various races to produce an average height which is representative of that group. A good example of this heritability factor is the gene HMGA2 (located at 12q14.3, chromosome 12). A 2007 study[3] found that a single letter change in that gene's coding is responsible for making some people taller than others.

Racial Differences in Height Caused by Specific Gene Differences

The scientists found that inheriting a form of the gene that has a "C" instead of a "T" written into it adds about half a centimeter (0.2 inches) to a person's height, while inheriting two copies adds nearly a full centimeter (0.4 inches). At least 25 percent of

3 "A common variant of HMGA2 is associated with adult and childhood height in the general population," Weedon, N. M., *Nature Genetics*, Oct. 2007; 39(10):1245–50.

Europeans carry two versions of the "tall" version of the gene, with another quarter carrying two "short" versions, a fact which accounts for height differentiation among Europeans.

In Peru, a 2018 study[4] among short indigenous people found an allele, of a gene called FBN1 (located at 15q21.1, chromosome 15) which differs by one base from the gene's usual DNA sequence. That change alters an amino acid in a carbohydrate-coated protein called fibrillin-1, and a person carrying just one copy of this variant is about 2.2 centimeters shorter than people with different versions of the gene. Those with two copies can be more than 4 centimeters shorter.

A 2022 study on genetics and height identified a total of 697 height-related genetic variants.[5] The study, an international collaboration by the Genetic Investigation of Anthropometric Traits (GIANT) consortium, analyzed nearly 5.4 million individuals of diverse ancestries. 75.8 percent of the analyzed sample were predominantly of European ancestry, 8.8 percent were predominantly East Asian, 8.5 percent were of Hispanic ethnicity, 5.5 percent, of African ancestry and 1.4 percent, of predominantly South Asian ancestry.

The 12,111 SNPs analyzed accounted for "40 percent (45 percent) of phenotypic variance in populations of European ancestry" and "around 10–20 percent (14–24 percent) in populations of other ancestries," the study said,[6] adding that "further research is needed to achieve equivalent saturation in other ancestries."

Physical Differences in Skeletal Structure as Used by Forensic Specialists and Anthropologists to Determine Race

Forensic scientists have long since known that it is possible to determine the race of an individual just by examining the skeletal structure.

4 "High altitude may have driven short stature in Peruvians," Pennisi, E., *Science,* 2018 May 18;360.
5 "A saturated map of common genetic variants associated with human height," Yengo, L., *Nature,* volume 610, pages 704–712 (2022).
6 *Ibid.*

The science is so accurate and detailed that it can detect race through any one of several race-specific skeletal characteristics:

— The presence of the oval window of the inner ear, which occurs more frequently in Whites than in Native Americans.

— The shape of the alveolar region which distinguishes between Asian, African, and North American Indian groups.

— The diameter of the neck.

— The height of the intercondylar notch (located at the end of the thigh bone that joins the knee).

— The femoral (thigh bone) curvature.

— The diameter and measurements of the pelvis.

— The measurements of the tibia (shinbone), and many others.[7] All of these traits will of course be genetic in origin, controlled by one or more of the genes listed above.

The Determination of Race by Skeletal Structure Alone

The advent of computer technology and Artificial Intelligence (AI) leapfrogged the evaluation process. In May 2022, for example, it was reported that "deep learning models based on artificial intelligence can identify someone's race just from their X-rays."[8]

An article in the *Science* journal revealed that scientists had trained their AI using hundreds of thousands of existing X-ray images labeled with details of the patient's race, an international team of health researchers from the US, Canada, and Taiwan tested their system on X-ray images that the computer software hadn't seen before (and had no additional information about). [9]

The AI could predict the reported racial identity of the patient on these images with surprising accuracy, even when the scans

7 "Determination of Race from the Skeleton through Forensic Anthropological Methods," Church. M.S., Forensic *Science Review,* 1995 Jun;7(1):1-39.
8 "AI Can Predict People's Race from X-Ray Images, And Scientists Are Concerned," *Science,* 18 May 2022.
9 *Ibid.*

were taken from people of the same age and the same sex. The system hit levels of 90 percent with some groups of images.[10]

A formal study on the ability of AI to detect race through x-rays alone added that ". . . the ability of AI deep learning models [to determine race] persisted over all anatomical regions and frequency spectrums of the images."[11]

Racial Skeletal Physical Differences Include Limb to Torso Ratios

These skeletal differences also play out in the ratio of limb length to torso length. As one paper from the UK's Oxford Brookes University put it, the "relationship between height and arm-span measurement in both sexes and different ethnic groups was studied in order to assess the use of the arm-span measurement as a suitable proxy indicator for height."[12]

The results, as spelled out in the paper, revealed that "arm-span was found to be significantly different from height in two ethnic groups, the Afro-Caribbeans of both sexes and Asian males.

"This suggests that any future studies attempting to use arm-span measurement as a proxy for height must consider these ethnic differences."[13]

Another older study, which presented the results of an anthropometric study of 293 Black and 298 White females, ages 35–89 years, "who were healthy, non-institutionalized residents of North Carolina,"[14] found marked differences between European Americans and African Americans, even bearing in mind

10 *Ibid.*

11 "AI recognition of patient race in medical imaging: a modelling study," The Lancet, Gichoya, J. W., et al., *The Lancet,* May 11, 2022

12 "The relationship between arm-span measurement and height with special reference to gender and ethnicity," Reeves, S. L., et al., School of Biological and Molecular Sciences, Oxford Brookes University, Headington, UK.PMID: 8793422.

13 *Ibid.*

14 "Arm-span, height, and age in black and white women," Steele, M.F., et al., *Annals of Human Biology*, 1990 Nov–Dec; 17(6):533-41

the fact that the latter have on average about 30 percent White ancestry.

Despite this, a review of the results found that the "correlation of arm-span and height for Black subjects was 0.852 and for White subjects was 0.903" and "age and arm-span correlation for White women was -0.167 and for Black women was -0.106."[15]

A 2014 study conducted in North Carolina had the stated aim of establishing "ratios between distinctive signature facial measurements such as head width and length, height and arm span, and forehead and lower face, and to compare them across gender and race."[16]

The study concluded that "after statistically analyzing the ratios, it was apparent that body measurements have a high correlation with racial background. It was concluded that the Golden Ratio is solely an ideal ratio that is not manifested within humans today; instead, it is seen prominently in nature, architecture, and art."

Racial Skeletal Differences in Bone Density and Strength

A 2016 study on the causes of male osteoporosis (the weakening of bones) carried out in Britain on European, African and South Asian men revealed a vast number of racial differences in areal bone mineral density (aBMD), volumetric bone mineral density (vBMD), bone geometry and strength.[17]

The study found that:[18]

— Africans had higher aBMD at the whole body, total hip and femoral neck compared to White and South Asian men independent of body size adjustments.

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15 *Ibid.*

16 "Correlation Between Body Measurements of Different Genders and Races," Goel, S., et al., Springer Proceedings in Mathematics and Statistics, (PROMS, volume 109), October 2014.

17 "Ethnic differences in bone geometry between White, Black and South Asian men in the UK," Zengin, A., *Bone*, 2016 Oct; 91: 180–185.

18 *Ibid.*

— Europeans had longer hip axis lengths than both African and South Asian men.

— Europeans also had "significantly lower cortical vBMD at the tibial diaphysis" [the long central portion of the tibia] than Africans or South Asians.

— At the diaphysis, South Asians have similar sized bones but thinner cortices than Europeans.

— At the tibia and radius diaphysis, Africans had larger bones with thicker cortices and greater bending strength than the other groups.

— At the metaphysis [the portion of the tibia just before the ennd of the bone], South Asians had smaller bones and lower trabecular vBMD [the partitions formed by bands of connective tissue].

— Africans had higher aBMD at the whole body, total hip and femoral neck than White men.

It should by now be needless to point out that all of these differences rely on individual genes, expressed in group format—evidence once again of the genetic, and thus biological, reality of race.[19]

19 As an aside to this chapter: The dominance of Africans—and particularly East Africans—in international athletics has led to the suggestion that there is a genetic basis to African athletic achievement. While on the face of it, this would most certainly be the most logical suggestion, it is worth noting that as of the time of writing (2022) there is no solid evidence for this claim. It is, however, as an article in the *British Medical Journal* said, "premature to argue that performance is not genetically mediated . . . This is largely due to limitations in the research method and capability to date, given small sample sizes and a focus on candidate gene polymorphisms . . . Future studies, using genome-wide association study methods, identifying a panel of single nucleotide polymorphisms known to affect physiology and performance, may further reveal the basis for advantage" ("The genetic basis for elite running performance," Tucker, R., et al., *British Journal of Sports Medicine*, 2013; 47:545–549).

CHAPTER NINE

THE GENES WHICH DETERMINE RACIAL DIFFERENCES IN INTELLIGENCE

The existence of differences in intelligence—in what are called "Intelligence Quotient" (IQ) scores— between the races has long been accepted by all as an established fact. The evidence for the existence of different scores in different parts of the world is so overwhelming that not even the race-denialists dare pretend that it does not exist.

Intelligence Quotient (IQ) Scores and Environment

Instead, the argument against the use of IQ scores as any sort of measurement is usually based on claims that "environment" plays a major role in determining intelligence.

This argument is, like *all* arguments against the tidal wave of genetic evidence, factually unsubstantiated. While it is certainly true that a deprived environment, such as a lack of suitable nutrition etc., can affect development, this can only ever be negative: it can never be positive. A "good" environment will only ever allow an individual to achieve their highest potential—but it will not change that highest potential level.

To give a practical example: a child with an IQ of 85, if sent to a higher institute of educational learning, under the best of circumstances, will never be able to achieve above his or her intelligence level. He or she will likely do "better" than in a negative environment, but the best possible circumstances in the world will not raise his or her IQ above 85.

IQ Scores by Race from Around the World

The debate about the "cause" of IQ differences is also flawed on account of the wealth of evidence showing that it is genetic in origin. Contrary to race-denialist claims, there is in fact a staggering amount of evidence showing that intelligence, brain size, and resultant ability is genetic—and that these IQ levels play out exactly according to the seven major racial categories already defined.

These IQ ranges can be summed up in the following table, arranged by race from those with the lowest average IQs to those with the highest:

Racial Group	Location	Average IQ	IQ Range
Bushmen	Southwestern	54	48–62
Aborigines	Australia	62	53–74
Aborigines	New Guinea	63	50–60
Sub-Saharan Africans	Africa[1]	67	59–89
Semites	Saudi Arabia[2]	84	84
Pacific Islanders	Pacific Islands	85	80–89
Mixed-race Africans[3]	United States	85	77–93
American Indians	North America	86	69–94
Southeast Asians (Burma, Thailand, Indonesia, Philippines, Malaysia)	S.E. Asia	87	85–93
Pacific Islanders	New Zealand	90	81–96
Arctic Peoples	North America	91	78–96
Europeans	Europe[6]	99	87–105
Europeans	United States[7]	101	96–109
Asians (China, Japan, and Korea)[8]	East Asia	105	100–120

Figures from *Race Differences in Intelligence: An Evolutionary Analysis,* Prof. Richard Lynn, Washington Summit Publishers, 2006; and *IQ and Global Inequality,* Profs. Richard Lynn and Tatu Vanhanen, Washington Summit Publishers, 2006.

Notes to this table:

[1] The statistics for North Africa are not included in the sub-Saharan figures. The reason for this is that North Africa has a mixed-race population of Semites, Europeans, and Africans. IQ scores for these nations are in the mid-80s range.

[2] DNA has shown that the Semitic race's heartland is the Saudi Arabian Peninsula. Jews, although carrying the Semitic founding gene, have mixed with other racial groups. The Ashkenazi Jews in particular, have on average 30 percent European admixture, which has dramatically boosted their IQ, in some instances to higher than the European average. Sephardic Jews, who have less outside admixture, consistently show an IQ level consistent with the Semitic average.

[3] DNA tests on Africans in the United States who have been there since the slavery era show European admixture of around 24 percent. This European ancestry has raised their IQ to above that of sub-Saharan Africans.

[4] American Indians have their ancestral origins in West Asia.

[5] Moaris have their origin in Southeast Asia and have since then mixed with Polynesians.

[6] There is considerable variation in IQ among Europeans, as the range (87 to 105) shows. The higher IQ levels (101 to 105) are concentrated in Northwest Europe, and lower IQ levels are in the South and the Southeast of Europe. These differing IQ levels in the latter regions are due to historical admixtures from outside Europe.

[7] The higher European IQ in America is because the majority of the US's founding white population were from Northwestern Europe. As the numbers of Northwestern Europeans has dropped

over the last decade due to increased immigration from other parts of Europe, this "original white American" IQ has steadily declined.

[8] This score is limited to selected groups within Southeast Asia and does not apply to all inhabitants of that region. Furthermore, it will be noted that the average Northwestern European IQ of 105 is equivalent to that of the average highest IQ in Southeast Asia.

Advances in Genetics Explain Racial IQ Differences

The starting point in understanding the relationship between race and IQ is that of physical brain size and shape. As is to be expected, brain sizes differ dramatically among the races.[1]

— The average cranial capacity of Southeast Asian males is 1,364 cm^3 (83.23 in^3).
— The average cranial capacity of European males is 1,347 cm^3 (82.19 in^3).
— The average cranial capacity of sub-Saharan African males is 1,267cm^3 (77.31 in^3).

Female readings in all cases were on average 100 cm^3 lower than for males. As with *all* "averages," it must be borne in mind that they are just that—and that in all sampled groups there will be significant numbers above and below the average reading.

Given these physical differences in cranial capacity, it is obvious that physical brain size must also differ. This is indeed the case, as numerous studies have shown.

The Genes which Determine Brain Size and Function

For example, a 2017 Chinese study[2] into brain size found that the CASC5 gene (also called KNL1, located at 15q15.1, chro-

1 "Brain size, IQ, and racial-group differences: Evidence from musculoskeletal traits," Rushton, J.P., et al., *Intelligence,* Volume 31, Issue 2, March–April 2003, Pages 139–155.

2 "Regional selection of the brain size regulating gene CASC5 provides new insight into human brain evolution," Shi, L., et al., *Human Genetics* 136 (2) 1–12, 2017.

mosome 15) protein had several amino acid mutations. Four of these mutations had "high frequencies of derived alleles" which only appear in East Asians, and which created the larger brain size. As the study put it, these alleles explain the "larger gray matter volume in East Asians."

A 2016 paper on the functioning of the neocortex area of the brain revealed that increases in the size of the brain are linked to higher cognitive ability.

The study said that the size of the neocortex is directly "related to an increase in the number of functionally distinct neocortical areas" and that the "neocortex in humans is widely regarded as the primary seat for the so-called higher cognitive functions, including self-awareness, consciousness, abstract reasoning and planning."[3]

In other words, the larger the cortex, the greater the cognitive ability. The cortex size is, of course, linked to the overall size of the brain.

A 2016 study also confirmed the definitive link between cranial capacity—that is, brain size—and intelligence.[4]

The paper, authored by an international team of scientists at universities in Britain, Australia, Germany, and the US, tested data from the UK Biobank for "shared genetic aetiology between cognitive functions and neuropsychiatric disorders and physical health."

The study found "highly significant associations were observed between the cognitive test scores in the UK Biobank sample and many polygenic profile scores, including . . . body mass index, intracranial volume, infant head circumference, and childhood cognitive ability."

3 "Neocortex expansion is linked to size variations in gene families with chemotaxis, cell-cell signalling and immune response functions in mammals," Atahualpa, C., et al., *Open Biology,* January 2016.

4 "Shared genetic aetiology between cognitive functions and physical and mental health in UK Biobank (N=112 151) and 24 GWAS consortia," S P Hagenaars et al, *Molecular Psychiatry,* volume 21, pages 1624–1632 (2016).

The paper concluded that "the genetic associations between infant head circumference and intracranial volume with educational attainment and verbal-numerical reasoning are important in themselves, as are many other cognitive–mental health and cognitive–physical health associations."[5]

The Genes Which Determine Intelligence

A 2012 study saw a global team of scientists identify the genes which control differences in brain size and intelligence.[6] They found that a variant in a gene called HMGA2 (located at 12q14.3, chromosome 12, and SNP rs10784502) affected brain size, as well as a person's intelligence.

As paper author Paul Thompson explained to the media,[7] DNA is comprised of four bases: A, C, T, and G. People whose HMGA2 gene held a letter "C" instead of "T" on that location of the gene possessed larger brains and scored more highly on standardized IQ tests.

"This is a really exciting discovery: that a single letter change leads to a bigger brain," said Thompson. "We found fairly unequivocal proof supporting a genetic link to brain function and intelligence. For the first time, we have watertight evidence of how these genes affect the brain. This supplies us with new leads on how to mediate their impact."[8]

In a 2017 study undertaken on 78,308 individuals,[9] the scientists found 336 associated SNPs in 18 genomic loci, "relevant to the creation of intelligence." Half of the SNPs so identified are located inside a gene, "implicating 22 genes," and further analysis detected another 30 genes which also play a role in shaping intel-

5 *Ibid.*

6 "Identification of common variants associated with human hippocampal and intracranial volumes," Stein, J. l., *Nature Genetics*, volume 44, pages 552–561 (2012).

7 "New genes linked to brain size, intelligence," *ScienceDaily,* April 15, 2012.

8 *Ibid.*

9 "Genome-wide association meta-analysis of 78,308 individuals identifies new loci and genes influencing human intelligence," Sniekers S., *Nature Genetics* Volume 49, No. 7, July 2017.

ligence. These genes are expressed in brain tissue, and "provide new insight into the genetic architecture of intelligence."[10] It added that the SNP with the "strongest emerging association with intelligence" is rs2490272 (located at 6q21, chromosome 6) in an intronic region of the gene FOXO3. The paper's Table 1, titled "Genomic loci and lead SNPs associated with intelligence" listed the 18 SNPS and their locations as follows:

Table 1

Genomic loci and lead SNPs associated with intelligence in the meta-analysis based on N=78,308.

rsID	Annotation	Locus[a]	Ref	Alt	RefF	Z	P-value	Direction[b]	N	N_{GWS}
rs2490272	FOXO3 intronic	6q21	t	c	0.63	7.44	9.96E-14	++++-+++	78307	28
rs9320913	intergenic	6q16.1	a	c	0.48	6.61	3.79E-11	++++-+++	78307	13
rs10236197	PDE1C intronic	7p14.3	t	c	0.63	6.46	1.03E-10	+++++-++	78286	35
rs2251499	intergenic	13q33.2	t	c	0.26	6.31	2.74E-10	++++++++	78307	22
rs36093924	CYP2D7 ncRNA_intr	22q13.2	t	c	0.46	−6.31	2.87E-10	?--?????	54119	100
rs7646501	intergenic	3p24.2	a	g	0.74	6.02	1.79E-09	?++-++++	65866	5
rs4728302	EXOC4 intronic	7q33	t	c	0.60	−5.97	2.42E-09	---+--+-	78307	45
rs10191758	ARHGAP15 intronic	2q22.3	a	g	0.61	−5.93	3.06E-09	?--?????	54119	17
rs12744310	intergenic	1p34.2	t	c	0.22	−5.88	4.20E-09	?------	65866	28
rs66495454	NEGR1 upstream	1p31.1	g	gtcct	0.62	−5.75	9.08E-09	?--?????	54119	1
rs113315451	CSE1L intronic	20q13.13	a	attat	0.43	5.71	1.15E-08	?+??????	54119	1
rs12928404	ATXN2L intronic	16p11.2	t	c	0.59	5.71	1.15E-08	++++++++	78307	19
rs41352752	MEF2C intronic	5q14.3	t	c	0.97	−5.68	1.35E-08	?--?????	54119	1
rs13010010	LINC01104 ncRNA_intr	2q11.2	t	c	0.38	5.65	1.56E-08	++++++++	78308	11
rs16954078	SKAP1 intronic	17q21.32	a	t	0.21	−5.55	2.84E-08	?----+--	65866	7
rs11138902	APBA1 intronic	9q21.11	a	g	0.54	5.49	4.12E-08	+++++-++	78307	1
rs6746731	ZNF638 intronic	2p13.2	t	g	0.43	−5.46	4.88E-08	-----+--	78307	1
rs6779302	intergenic	3p24.3	t	g	0.37	−5.45	4.99E-08	?--?????	54119	1

10 *Ibid.*

A 2018 study saw several leading behavioral geneticists confirm that intelligence is genetic—and that IQ DNA test kits will soon be available in the same way that there are already ancestry DNA tests.[11]

As reported by the Massachusetts Institute of Technology's *MIT Technology Review,* "gene studies have finally gotten big enough—and hence powerful enough—to zero in on genetic differences linked to IQ."

More than five hundred genes have now been tied to performance on IQ tests, and "results from an experiment correlating one million people's DNA with their academic success are due at any time."

Quoting Robert J. Plomin, an American psychologist and geneticist best known for his work in twin studies and behavior genetics, the *MIT Technology Review* said that the new discoveries "mean we can now read the DNA of a young child and get a notion of how intelligent he or she will be."

Plomin outlined the DNA IQ test scenario in January 2018 in a paper titled, "The New Genetics of Intelligence,"[12] making a case that parents will use direct-to-consumer tests to predict children's mental abilities and make schooling choices, a concept he calls precision education.

The implications of all these studies—and there are many more—is that *genes determine brain size, structure, and intelligence.*

Two questions must now be answered:

1. How exactly do these specific genes "create intelligence?" and

2. How are the genes "different" in the races so that they produce the IQ differences outlined earlier in this chapter?

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11 "DNA tests for IQ are coming, but it might not be smart to take one," Antonio Regalado, *MIT Technology Review*, April 2, 2018.

12 "The new genetics of intelligence," Robert Plomin & Sophie von Stumm, *Nature Reviews Genetics,* volume 19, pages 148–159, January 8, 2018.

Racial Differences in Genetics Which Cause Racial Differences in Intelligence

The answer to both these questions can be found in an important 2018 study,[13] and the comparison of that study's results with the databases of "The 1000 Genomes Project."[14]

This latter project, which was first created in 2008 and completed its work in 2015, was an international research effort to establish a detailed catalog of human genetic variation.

"The 1000 Genomes Project" uses an allied database viewer, known as "Ensembl," to allow users to get graphical representations of SNPs and their frequencies in five different population groups, Africans, American (Amerindian), East Asian, European, and South Asian.[15]

The 2018 study was conducted by an international team of scientists from the United States, the Netherlands, Austria, Australia, Belgium, Italy, Finland, the United Kingdom, Germany, Switzerland, Saudi Arabia, Iceland, Greece, Sweden, China, Estonia, Singapore, Denmark, Qatar, France; teams from the DNA testing company 23andMe, and the COGENT (the Cognitive Genomics Consortium).

Together, this large group was able to draw together DNA from over 1.1 million test subjects from around the globe. The scientists then undertook a laborious "large-scale genetic association analysis of educational attainment" and ability, using data from 23andMe on self-reported ability in mathematics and other high cognitive activities.[16] By comparing the SNP analysis of everyone with their educational attainment and cognitive ability,

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13 "Gene discovery and polygenic prediction from a genome-wide association study of educational attainment in 1.1 million individuals," Lee, J.J., *Nature Genetics,* volume 50, pages 1112–1121 (2018).

14 "The 1000 Genomes Project" and its databases can be found online at internationalgenome.org.

15 The gene browser associated with "The 1000 Genomes Project," called "Ensembl," can be found online at ensembl.org.

16 Lee, J.J., *Nature Genetics op. cit.*

the scientists were able to identify a list of "genome-wide-significant SNPs" in both genes and X-chromosomes, all which are "involved in brain-development processes and neuron-to-neuron communication."[17]

In total, the scientists identified 1,281 SNPs, including 10 in the X-chromosome, all of which were common to the successful and intelligent subjects in their dataset.

From here, they next were able to generate an equation which would, just by using the identified SNPS, predict whether any individual would have a high intelligence or otherwise. The SNPs were divided into categories such as "366 independent SNPs that reached genome-wide significance of highest math class," and so on.

As the scientists were able to identify specific SNPs, they were also able to report on exactly which nucleobases (Adenine, "A"; Cytosine, "C"; Guanine, "G"; and Thymine, "T") were involved in the SNP's reaction.

(It will be recalled from Chapter Two of this book that SNPs are the points of connection between the two sides of the "twisted ladder" helix) of DNA.

In this way, for example, one of the SNPs linked to advanced mathematical ability, rs9556702[18] (found in the MBNL2 gene, located at 13:97337467, chromosome 13) is noted by a linking of the A and G nucleobases.

These SNPs are divided into "A" and "G" type alleles. If an individual has the "A" type allele (in this case, a "nitron variant"), they are likely to have a higher mathematical ability—and anyone who has the "G" type allele will not be so advanced in mathematical ability.

17 *Ibid.*

18 Supplementary Table 11. Association results for the 618 independent SNPs that reached genome-wide significance (P < 5×10-8) in GWAS of math ability (self-reported), in "Gene discovery and polygenic prediction from a genome-wide association study of educational attainment in 1.1 million individuals," Lee, J.J., *Nature Genetics op. cit.*

Distribution of the "A" allele SNP nitron variant rs9556702 among five races, as demonstrated by The 1000 Genome Consortium's "Ensembl" gene browser. Key to the pie graphs: AFR =African, AMR=American (Amerindian), EAS = East Asians, EUR=Europeans, SAS=South Asians.

The same scenario plays out (with different nucleobases of course) in every other SNP identified in the study.

Intelligence Genes Differ by Racial Distribution

According to the Ensembl gene browser, the "A" allele of the rs9556702 SNP is found among only 36 percent of the world's population.

This is a small number, but when broken down into the five racial groups used by Ensembl, a dramatically different picture emerges:

Africans — only 7 percent have the "A" allele.

Americans (Amerindian) — only 38 percent have the "A" allele.[19]

East Asians — 49 percent have the "A" allele.

Europeans — 53 percent have the "A" allele.

South Asians — 49 percent have the "A" allele.

Put another way: the "A" allele nitron variant is 178 times more likely to be found in a European than an African, and at an only slightly lower ratio in Asian and related populations.

The implications of the unequal distribution of intelligence-related SNPs are weighty: it means that racial groups who have these gene at higher ratios will, on average, have a higher intelligence than those who do not.

List of the Most Important Genes Affecting Intelligence—and Their Unequal Distribution Among the Races

Here follows a table of SNPs which have definitively been linked to brain function, as detailed in the 2018 *Nature Genetics* study listed above. These SNPS have then been cross correlated with frequencies in three racial groups as defined by "The 1000 Genome Consortium" and in the Ensembl machine.

19 It should be recalled that present day Amerindians not only originated in East Asia, but also have European ancestry.

SNP	% European	% African	% European vs African	% Asian	% Asian vs African	Percentage by which Europeans are *more likely* to have this gene than Africans
rs8056960(C)	56 %	13 %	336 %	81 %	527 %	336 %
rs9309922(T)	81 %	39 %	108 %	100 %	158 %	108 %
rs9328534(T)	68 %	25 %	176 %	57 %	130 %	176 %
rs9387711(T)	57 %	24 %	138 %	47 %	95 %	139 %
rs9388490(T)	44 %	20 %	121 %	98 %	392 %	121 %
rs9397806(A)	35 %	17 %	113 %	30 %	78 %	113 %
rs9513780(T)	47 %	13 %	261 %	36 %	172 %	261 %
rs9556702(A)	53 %	3 %	1641 %	49 %	1516 %	1639 %
rs9607805(T)	70 %	30 %	136 %	43 %	44 %	136 %
rs9611186(G)	61 %	28 %	119 %	82 %	194 %	119 %
rs9682919(A)	64 %	19 %	233 %	75 %	293 %	233 %
rs9889827(T)	68 %	23 %	201 %	59 %	160 %	201 %
rs9933476(G)	52 %	15 %	260 %	74 %	409 %	260 %
rs10057590(A)	47 %	18 %	164 %	27 %	49 %	164 %
rs10078630(A)	56 %	11 %	388 %	42 %	265 %	389 %
rs10124571(T)	38 %	10 %	286 %	43 %	337 %	286 %
rs10205421(G)	35 %	8 %	337 %	29 %	263 %	338 %
rs10233848(A)	68 %	28 %	142 %	46 %	63 %	142 %
rs10401883(T)	69 %	34 %	106 %	44 %	30 %	106 %
rs10402747(T)	53 %	16 %	227 %	88 %	440 %	227 %
rs10411958(T)	49 %	20 %	144 %	32 %	60 %	144 %
rs10432638(A)	35 %	3 %	1272 %	65 %	2439 %	1274 %
rs10476217(G)	83 %	31 %	163 %	48 %	51 %	163 %
rs10740140(A)	46 %	11 %	316 %	57 %	416 %	316 %
rs10786832(T)	40 %	14 %	195 %	70 %	415 %	196 %
rs10821080(T)	49 %	9 %	439 %	52 %	471 %	439 %
rs10821977(A)	55 %	8 %	558 %	82 %	887 %	559 %
rs10863246(A)	35 %	7 %	387 %	57 %	699 %	387 %
rs10889949(T)	57 %	26 %	115 %	67 %	154 %	115 %
rs10953137(G)	47 %	20 %	130 %	39 %	94 %	130 %
rs11125721(A)	39 %	3 %	1434 %	15 %	471 %	1437 %
rs11192147(A)	47 %	16 %	188 %	66 %	308 %	188 %

SNP	% European	% African	% European vs African	% Asian	% Asian vs African	Percentage by which Europeans are *more likely* to have this gene than Africans
rs11210394(T)	35 %	3 %	960 %	72 %	2066 %	960 %
rs11576565(A)	52 %	4 %	1132 %	35 %	719 %	113 %
rs11685902(T)	53 %	13 %	318 %	42 %	228 %	318 %
rs11768845(T)	63 %	20 %	210 %	42 %	110 %	210 %
rs11775314(T)	36 %	7 %	442 %	50 %	667 %	443 %
rs11793831(T)	43 %	10 %	350 %	48 %	397 %	350 %
rs11864066(G)	57 %	11 %	420 %	78 %	610 %	421 %
rs12136092(A)	61 %	7 %	799 %	35 %	413 %	799 %
rs12146618(T)	42 %	9 %	358 %	24 %	163 %	359 %
rs12208753(A)	54 %	21 %	158 %	45 %	112 %	158 %
rs12404086(T)	60 %	7 %	798 %	35 %	415 %	797 %
rs12491181(T)	46 %	5 %	830 %	46 %	827 %	830 %
rs12548560(A)	71 %	35 %	100 %	77 %	116 %	100 %
rs12567360(A)	47 %	17 %	183 %	49 %	194 %	183 %
rs12701207(T)	65 %	26 %	154 %	47 %	83 %	154 %
rs12712784(T)	39 %	5 %	718 %	6 %	27 %	718 %
rs12773994(T)	62 %	30 %	107 %	12 %	-59 %	107 %
rs12799722(A)	37 %	15 %	147 %	21 %	40 %	148 %
rs12814021(T)	55 %	19 %	187 %	52 %	172 %	187 %
rs12919084(T)	48 %	14 %	237 %	37 %	163 %	236 %
rs13004882(T)	43 %	2 %	2166 %	17 %	794 %	2165 %
rs13019313(A)	61 %	25 %	145 %	39 %	56 %	145 %
rs13071190(T)	69 %	18 %	289 %	47 %	165 %	289 %
rs13116036(T)	47 %	2 %	2713 %	6 %	286 %	2719 %
rs13270346(A)	34 %	2 %	2173 %	9 %	488 %	2178 %
rs13413443(T)	38 %	3 %	1392 %	9 %	250 %	1394 %
rs13428598(T)	37 %	7 %	415 %	0 %	-97 %	417 %
rs16950305(G)	83 %	42 %	100 %	92 %	120 %	100 %
rs17048855(A)	35 %	5 %	595 %	47 %	847 %	595 %
rs17106817(T)	72 %	29 %	145 %	91 %	211 %	145 %
rs17266097(T)	39 %	3 %	1217 %	18 %	507 %	1218 %
rs17608413(T)	45 %	22 %	109 %	47 %	116 %	109 %

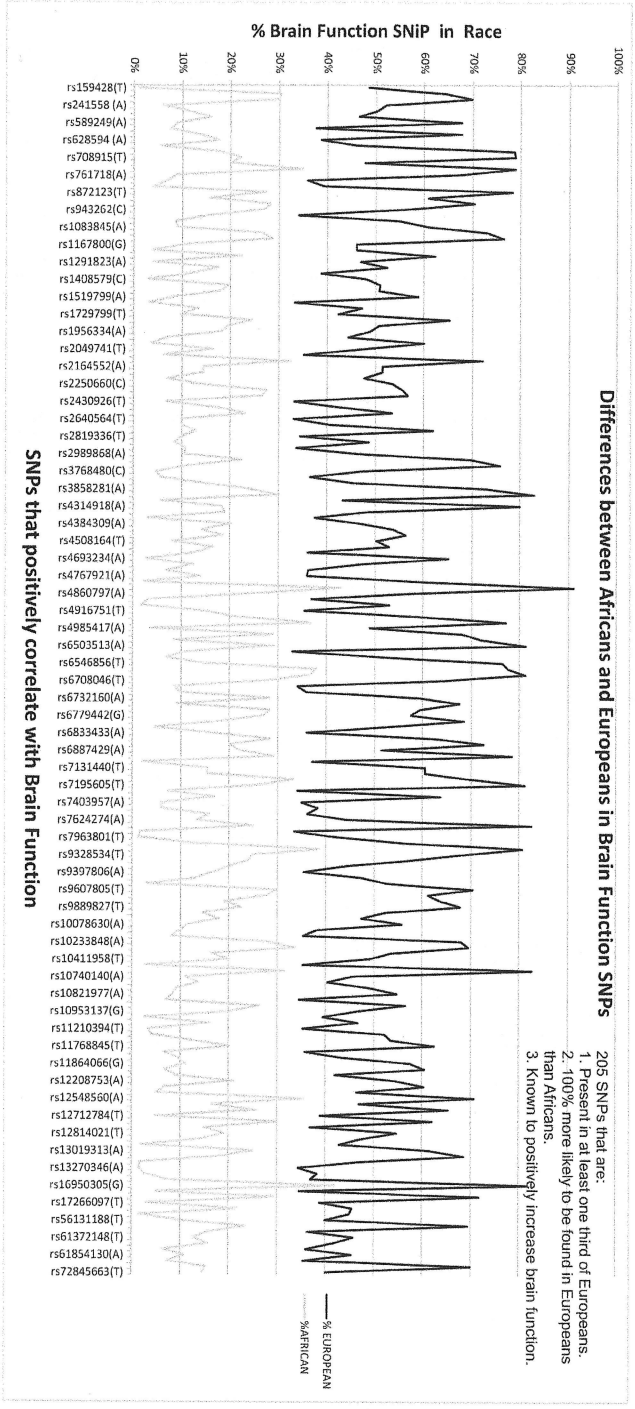
SNP	% European	% African	% European vs African	% Asian	% Asian vs African	Percentage by which Europeans are *more likely* to have this gene than Africans
rs34389480(A)	45 %	2 %	2600 %	20 %	1124 %	2605 %
rs56131188(T)	40 %	10 %	306 %	0 %	-99 %	307 %
rs56146203(A)	69 %	24 %	195 %	60 %	156 %	195 %
rs59692040(T)	36 %	16 %	129 %	25 %	60 %	130 %
rs61372148(T)	46 %	13 %	260 %	59 %	360 %	260 %
rs61527214(A)	42 %	16 %	163 %	64 %	304 %	164 %
rs61816194(T)	36 %	6 %	466 %	15 %	135 %	467 %
rs61854130(A)	45 %	11 %	319 %	58 %	440 %	319 %
rs62383977(T)	35 %	7 %	387 %	43 %	487 %	388 %
rs66493370(A)	70 %	15 %	358 %	47 %	209 %	358 %
rs72845663(T)	40 %	15 %	174 %	22 %	54 %	176 %

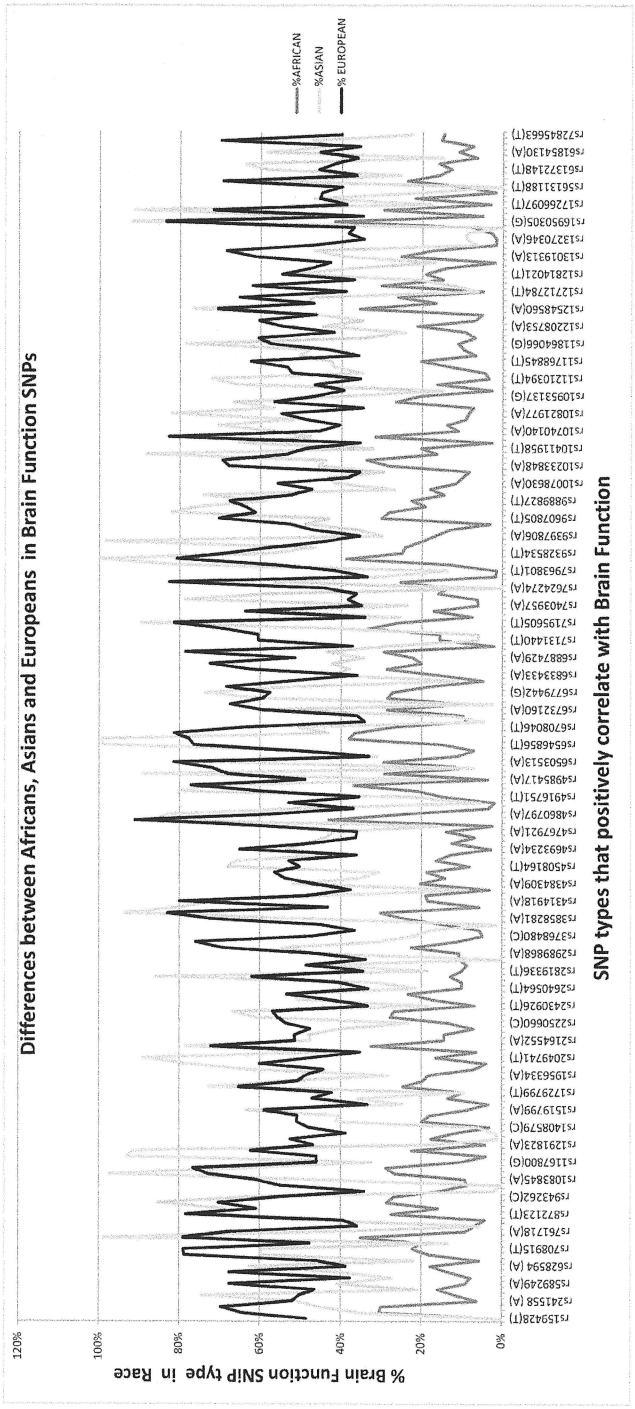
Note: This is only a partial list.

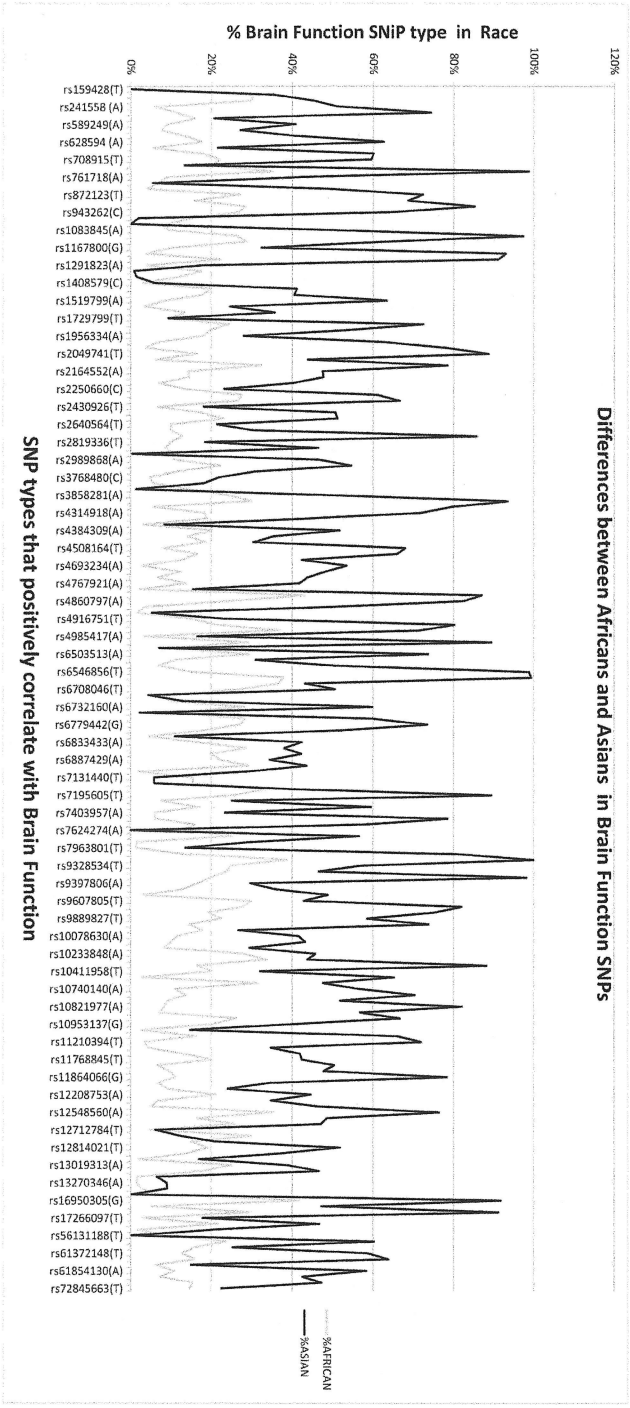
On the following three pages, some of these results above are presented in graph format. From that, the differences in intelligence SNP distribution become even more obvious:

There are numerous other examples of this widely varying distribution of intelligence-linked genes, SNPs, and alleles, all of which match perfectly with the IQ scores as outlined at the beginning of this chapter.

It is likely, as time goes on, that even further genetic links to intelligence, and the differentiation according to race, will emerge, but the scientific evidence is already clear and irrefutable that intelligence is genetic, and therefore inherited.


% Brain Function SNiP in Race
Differences between Africans and Europeans in Brain Function SNPs
205 SNPs that are:
1. Present in at least one third of Europeans.
2. 100% more likely to be found in Europeans than Africans.
3. Known to positively increase brain function.
SNPs that positively correlate with Brain Function
% EUROPEAN
%AFRICAN
0%
10%
20%
30%
40%
50%
60%
70%
80%
90%
100%
rs159428(T)
rs241558 (A)
rs589249(A)
rs628594 (A)
rs708915(T)
rs761718(A)
rs872123(T)
rs943262(C)
rs1083845(A)
rs1167800(G)
rs1291823(A)
rs1408579(C)
rs1519799(A)
rs1729799(T)
rs1956334(A)
rs2049741(T)
rs2164552(A)
rs2250660(C)
rs2430926(T)
rs2640564(T)
rs2819336(T)
rs2989868(A)
rs3768480(C)
rs3858281(A)
rs4314918(A)
rs4384309(A)
rs4508164(T)
rs4693234(A)
rs4767921(A)
rs4860797(A)
rs4916751(T)
rs4985417(A)
rs6503513(A)
rs6546856(T)
rs6708046(T)
rs6732160(A)
rs6779442(G)
rs6833433(A)
rs6887429(A)
rs7131440(T)
rs7195605(T)
rs7403957(A)
rs7624274(A)
rs7963801(T)
rs9328534(T)
rs9397806(A)
rs9607805(T)
rs9889827(T)
rs10078630(A)
rs10233848(A)
rs10411958(T)
rs10740140(A)
rs10821977(A)
rs10953137(G)
rs11210394(T)
rs11768845(T)
rs11864066(G)
rs12208753(A)
rs12548560(A)
rs12712784(T)
rs12814021(T)
rs13019313(A)
rs13270346(A)
rs16950305(G)
rs17266097(T)
rs56131188(T)
rs61372148(T)
rs61854130(A)
rs72845663(T)


Differences between Africans, Asians and Europeans in Brain Function SNPs
%AFRICAN
%ASIAN
% EUROPEAN
% Brain Function SNP type in Race
120%
100%
80%
60%
40%
20%
0%
SNP types that positively correlate with Brain Function
rs1594428(T)
rs2415S8 (A)
rs5892491A)
rs628594 (A)
rs7089151T)
rs761718(A)
rs872123(T)
rs943262(C)
rs10838451A)
rs11678001G)
rs1291823(A)
rs1408579(C)
rs1519799(A)
rs1729799(T)
rs1956334(A)
rs2049741(T)
rs2164552(A)
rs2250660(C)
rs2430926(T)
rs2640564(T)
rs2819336(T)
rs2898868(A)
rs3768480(C)
rs3858281(A)
rs4314918(A)
rs4384309(A)
rs4508164(T)
rs4693234(A)
rs4767921(A)
rs4860797(A)
rs4916751(T)
rs4985417(A)
rs5503513(A)
rs5546856(T)
rs6708046(T)
rs6732160(A)
rs6779442(G)
rs6833433(A)
rs6887429(A)
rs7131440(T)
rs7195605(T)
rs7403957(A)
rs7624274(A)
rs7963801(T)
rs9328534(T)
rs9397806(A)
rs9607805(T)
rs9889827(T)
rs10078630(A)
rs10233848(A)
rs10411958(T)
rs10740140(A)
rs10821977(A)
rs10953137(G)
rs11211039(T)
rs11768845(T)
rs11864066(G)
rs12208753(A)
rs12548560(A)
rs12712784(T)
rs12814021(T)
rs13019313(A)
rs13270346(A)
rs16950305(G)
rs17266097(T)
rs5613118B(T)
rs6137214B(T)
rs6185413O(A)
rs7284566(T)


% Brain Function SNiP type in Race
0%
20%
40%
60%
80%
100%
120%
rs159428(T)
rs241558 (A)
rs589249(A)
rs628594 (A)
rs708915(T)
rs761718(A)
rs872123(T)
rs943262(C)
rs1083845(A)
rs1167800(G)
rs1291823(A)
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rs1519799(A)
rs1729799(T)
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rs2164552(A)
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rs2430926(T)
rs2640564(T)
rs2819336(T)
rs2989868(A)
rs3768480(C)
rs3858281(A)
rs4314918(A)
rs4384309(A)
rs4508164(T)
rs4693234(A)
rs4767921(A)
rs4860797(A)
rs4916751(T)
rs4985417(A)
rs6503513(A)
rs6546856(T)
rs6708046(T)
rs6732160(A)
rs6779442(G)
rs6833433(A)
rs6887429(A)
rs7131440(T)
rs7195605(T)
rs7403957(A)
rs7624274(A)
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rs10740140(A)
rs10821977(A)
rs10953137(G)
rs11210394(T)
rs11768845(T)
rs11864066(G)
rs12208753(A)
rs12548560(A)
rs12712784(T)
rs12814021(T)
rs13019313(A)
rs13270346(A)
rs16950305(G)
rs17266097(T)
rs56131188(T)
rs61372148(T)
rs61854130(A)
rs72845663(T)
SNP types that positively correlate with Brain Function
Differences between Africans and Asians in Brain Function SNPs
%AFRICAN
%ASIAN

CHAPTER TEN

THE GENES WHICH DETERMINE RACIAL DIFFERENCES IN PSYCHOLOGY

According to the American Psychological Association, "psychologists examine the relationships between brain function and behavior, and the environment and behavior, applying what they learn to illuminate our understanding and improve the world around us."[1]

The important part of that definition, for the purposes of this study, is the "relationship between brain function and behavior."

As discussed in the previous chapter, the human brain's physical structure and size varies by race and these differences are genetic in origin.

As the brain works by sending and receiving chemical and electrical signals throughout the body, it is obvious that its structure and chemical composition is an intrinsic part of its operations—and because these attributes are physical in origin and inherited, they will be affected by genes as well.

This in turn will affect behavior and "psychology"—both of these attributes only being the product of the gene-determined chemical reactions in the brain. It should therefore come as no surprise—even if race-denialists hotly contest it—that race will play a role in behavior, or psychology, as well.

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1 Science of Psychology, American Psychological Association, apa.org/education-career/guide/science.

The Genetic Basis of Schizophrenia

One of the best examples of this is the mental illness known as schizophrenia.

According to the official definition as issued by the US government's National Institute of Mental Health (NIMH), schizophrenia "is a serious mental disorder in which people interpret reality abnormally. Schizophrenia may result in some combination of hallucinations, delusions, and extremely disordered thinking and behavior that impairs daily functioning, and can be disabling."[2]

In addition, the NIMH said the cause of schizophrenia lies in the "differences in brain structure, function, and interactions among chemical messengers (called neurotransmitters)."

In particular, the NIMH continues, "differences in the volumes of specific components of the brain, in the way regions of the brain are connected and work together, and in neurotransmitters, such as dopamine, are found in people with schizophrenia."[3] More significantly, the NIMH says that psychotic episodes are most common in "people who are vulnerable due to genetics."

Until the advent of modern genetic studies, it was known only that schizophrenia appeared to be inherited, or "ran in families." However, the genetic component of schizophrenia was confirmed in 2014 with a report in the *Molecular Psychiatry* journal:

"It has long been recognized that generalized deficits in cognitive ability represent a core component of schizophrenia (SCZ), evident before full illness onset and independent of medication.[4]

"The possibility of genetic overlap between risk for SCZ and cognitive phenotypes has been suggested by the presence of

2 "Schizophrenia," National Institute of Mental Health (NIMH), May 2020.
3 *Ibid.*
4 "Molecular genetic evidence for overlap between general cognitive ability and risk for schizophrenia: a report from the Cognitive Genomics consorTium (COGENT)," T Lencz, at al., *Molecular Psychiatry,* Feb. 2014; 19(2):168–74.

cognitive deficits in first-degree relatives of patients with SCZ; however, until recently, molecular genetic approaches to test this overlap have been lacking. Within the last few years, large-scale genome-wide association studies (GWAS) of SCZ have demonstrated that a substantial proportion of the heritability of the disorder is explained by a polygenic component consisting of many common single-nucleotide polymorphisms (SNPs) of extremely small effect."[5]

A 2017 study published in the *Schizophrenia Bulletin* also pointed out that there "is evidence for genetic overlap between cognitive abilities and schizophrenia (SCZ), and genome-wide association studies (GWAS) demonstrate that both SCZ and general cognitive abilities have a strong polygenic component with many single-nucleotide polymorphisms (SNPs) each with a small effect."[6]

A 2018 study, published in the *Scandinavian Journal of Psychology,* pointed this out in no uncertain terms, along with the clear identification of genes linked to intelligence.[7] The study reported that genome-wide association studies (GWAS) had identified "100 risk gene loci," and that "overlapping gene loci were found between education and SCZ, and between SCZ and cognitive traits, suggesting common genetic risk between SCZ and cognitive dysfunction."

The paper concluded by saying that the "evidence suggests a polygenic architecture for SCZ and cognitive functions, and a large degree of shared genetic risk."[8]

5 *Ibid.*

6 "Identification of Gene Loci That Overlap Between Schizophrenia and Educational Attainment," Hellard, S. L. H., et al., *Schizophrenia Bulletin,* May 1, 2017;43(3):654–664.

7 This study added that the genome-wide association studies it reviewed also revealed "more than 20 gene loci associated with cognitive traits, including intelligence, general cognition (g-factor), reaction time and verbal-numerical reasoning," and that "several gene loci have been implicated in educational attainment, a proxy measure of cognitive function." Hellard, S. L. H., et al., *Schizophrenia Bulletin, op. cit.*

8 "How can genetics help understand the relationship between cognitive

Genetic Link between Schizophrenia, Low Intelligence, and Criminal Activity

In other words, all the scientific research shows a clear link between schizophrenia and low intelligence (cognitive dysfunction). There is also a link between schizophrenia and criminal activity. This is not to say that all schizophrenics are criminals, but among criminals, the incidence of schizophrenia is disproportionately high.

As early as 1986, scientists had already clearly linked schizophrenia to violent crime.[9] A 2008 study specified what sorts of crimes are notable among schizophrenics:

"There is now robust evidence demonstrating that both men and women with schizophrenia are at elevated risk when compared to the general population to be convicted of non-violent criminal offences, at higher risk to be convicted of violent criminal offences, and at even higher risk to be convicted of homicide."[10]

A 2009 report in the *Journal of the American Medical Association* revealed that "persons with schizophrenia are thought to be at increased risk of committing violent crime 4 to 6 times the level of general population individuals without this disorder."[11] That study also specified the sorts of crimes involved: homicide, assault, robbery, arson, any sexual offense, illegal threats, and intimidation.[12]

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dysfunction and schizophrenia?" Olav B Smeland, Ole A Andreassen, *Scandinavian Journal of Psychology,* Feb. 2018; 59(1):26–31.

9 "Relationships between dimensional factors of psychopathy and schizotypy," Ragsdale KA, Bedwell JS, *Frontiers in Psychology,* Jul. 26, 2013; 4:482.

10 "Violent behaviour among people with schizophrenia: a framework for investigations of causes, and effective treatment, and prevention," Sheilagh Hodgins, *Philosophical Transactions of the Royal Society B: Biological Sciences,* Aug. 12, 2008; 363(1503): 2505–2518.

11 "Schizophrenia, Substance Abuse, and Violent Crime," Seena Fazel, et al., *Journal of the American Medical Association,* May 20, 2009; 301(19): 2016–2023.

12 *Ibid.*

The relationship has been confirmed in numerous studies since then,[13] and perhaps most dramatically in a 2013 overview of all the available literature, which concluded as follows:

"The main goal of this review was to provide clinicians with a critical summarizing of all major relevant studies published during 15 years. It is concluded that major mental disorders per se, especially schizophrenia, even without alcohol or drug abuse, are indeed associated with higher risks for interpersonal violence."[14]

A 2014 study in Israel found that "increased risk of violence is part of the clinical picture of schizophrenia and needs to be recognized as a legitimate, essential, aspect of clinical management."[15]

Another 2014 study added that there "is now robust evidence that schizophrenia is associated with an increased risk of violence. Across Europe, the numbers of forensic hospital beds have dramatically increased largely due to admissions of men with schizophrenia . . . People with schizophrenia are at increased risk, as compared to the general population, to be convicted for violent crimes because they are more likely to engage in aggressive behaviour towards others."[16]

A 1998 study showed that there "is a real link between violence and crime on one hand and mental illness including schizophrenia on the other hand. This link persists even after controlling for demographic and socio-economic variables; controlling for these factors did not cause the relationship between crime and mental

13 "Mental disorder and crime. Evidence from a Danish birth cohort," S Hodgins et al., *General Psychiatry,* June 1996; 53(6):489–96; and "Major mental disorders and violence: a critical update," Joyal C. C. et al., *Current Psychiatry Reviews,* Volume 3, Issue 1, 33–50, 2007.

14 "Relationships between dimensional factors of psychopathy and schizotypy," Ragsdale KA, Bedwell JS, *Frontiers in Psychology,* July 26, 2013; 4:482.

15 "Schizophrenia and violent crime: a population-based study," A Fleischman et al., *Psychological Medicine,* Oct. 2014; 44(14):3051–7.

16 "Schizophrenie und Gewalt," S Hodgins, R Müller-Isberner, *Der Nervenarzt,* volume 85, pages 273–278 (March 2014).

illness to disappear. . . The probability of mentally ill patients to be criminal or violent is increased by their use of alcohol and other psychoactive substances."[17]

Another study on the same topic showed that the incidence of violent crime increases by a factor of 30 percent (from 21.3 to 30.33) in individuals "comorbid for both schizophrenia and substance abuse."[18]

The Specific Genetic Basis of Schizophrenia

Although it was long known that schizophrenia—like all mental illnesses—was inherited, it was only with the emergence of the DNA era that it was tracked down to specific genes.

A 2019 report on the topic[19] revealed that a global consortium called "Schizophrenia Exome Sequencing Meta-Analysis" had gathered data on schizophrenic patients' exomes (the DNA that codes for protein). After years of research, 10 variant genes (called disabled types) had been identified which were clearly linked to schizophrenia.

Like all genes, they had to have been present in the parents of an individual to be passed on: if one of a person's two copies of any of these genes are the disabled type, their risk of developing schizophrenia increased up to 50 times. Two genes, GRIN2A (located at 16p13.2, chromosome 16) and SP4 (located at 7p15.3, chromosome 7) were shown in particular to be markers for the disease.

In addition, deletions or duplications of genetic material in any of several chromosomes, which can affect multiple genes, also increase schizophrenia risk. In particular, a deletion in a region of chromosome 22 called 22q11 has been identified as

17 "Criminal and violent behavior in schizophrenic patients: an overview," J Modestin, *Psychiatry and Clinical Neurosciences,* Dec. 1998; 52(6):547–54.

18 "Clinical correlates of later violence and criminal offences in schizophrenia," Soyka M, Graz C, Bottlender R, Dirschedl P, Schoech H., *Schizophrenia Research,* Aug. 2007; 94(1-3):89–98.

19 "Rare genetic variants could point to new treatments for severe psychiatric disorder," Kaiser, J., Science, Oct. 25, 2019.

causing schizophrenia. Some individuals with this deletion have other features in addition to schizophrenia, such as heart abnormalities, immune system problems, and an opening in the roof of the mouth (cleft palate) and are diagnosed with a condition called 22q11.2 deletion syndrome.

Other variant genes identified earlier as contributing toward schizophrenia include AKT1[20] (located at 14q32.33, chromosome 14), COMT[21] (located at 22q11.21, chromosome 22), and the two C4 genes (located at 6p21.3, chromosome 6).[22]

In summary, the scientific evidence on schizophrenia shows the following:

1. Schizophrenia is a serious mental illness with a genetic basis;

2. There is a clear link between schizophrenia, low intelligence, and violent criminality;

3. Violent crime among schizophrenics is aggravated by substance abuse.

These are clear behavioral issues which can cause significant disruption in society.

Racial Differences in Schizophrenia Rates

Given all the above facts, it is disturbing to find out that non-Europeans, and Africans in particular, display a markedly higher rate of schizophrenia than do Europeans. In 2007, the *International Journal of Epidemiology* pointed out that immigrant "groups in Western Europe have markedly increased rates of

20 "AKT1 is associated with schizophrenia across multiple symptom dimensions in the Irish Study of High Density Schizophrenia Families (ISHDSF)," Thiselton, D. L., *Biological Psychiatry*, Mar. 1, 2008; 63(5): 449–457.

21 "COMT genetic variation confers risk for psychotic and affective disorders: a case control study," Funke, B., *Behavioral and Brain Functions* volume 1, Article number: 19 (2005).

22 "Overexpression of schizophrenia susceptibility factor human complement C4A promotes excessive synaptic loss and behavioral changes in mice," Yilmaz M., *Nature Neuroscience*, Feb. 2021; 24(2):214–224, and "Schizophrenia risk from complex variation of complement component 4," Sekar, A., *Nature* volume 530, pages 177–183 (2016).

schizophrenia. The highest rates are found in ethnic groups that are predominantly Black."[23]

That report went on to reveal that Blacks in the US are 200 percent more likely to develop schizophrenia than Whites, and that the "data indicate[s] substantially elevated rates of schizophrenia among African Americans in comparison with Whites in this birth cohort," and that this "variance" has also been noted in Britain.

A 2004 study, published in the journal *Administration and Policy in Mental Health and Mental Health Services Research,* studied the relationship "among race, diagnoses of schizophrenia, mood disorder, and admission to state psychiatric hospitals" in a sample of 2,311 individuals who had hospital admissions in Indiana during an 8-year period.[24]

"In comparison with the general population, African Americans were found to be overrepresented among inpatients with diagnoses of schizophrenia. African Americans were four times more likely than White inpatients to receive a diagnosis of schizophrenia," the study concluded.[25]

A 2014 study published in the *World Journal of Psychiatry,* based on a comprehensive summary of empirical research on race and diagnosis of psychotic disorders spanning a 24-year period, found that "African American/Black consumers show a rate [of schizophrenia] of on average three to four higher than Euro-American/White consumers."[26] In addition, the study added, "Latino American/Hispanic consumers were

23 "Race and risk of schizophrenia in a US birth cohort: another example of health disparity?" *International Journal of Epidemiology,* Aug. 2007; 36 (4): 751–8.

24 "Race, Schizophrenia, and Admission to State Psychiatric Hospitals," Arnold Barnes, *Administration and Policy in Mental Health and Mental Health Services Research* volume 31, pages 241–252 (2004).

25 *Ibid.*

26 "Racial disparities in psychotic disorder diagnosis: A review of empirical literature," Robert C Schwartz and David M Blankenship, *World Journal of Psychiatry,* Dec. 22, 2014; 4(4): 133–140.

also disproportionately diagnosed with psychotic disorders on average approximately three times higher compared to Euro-American/White consumers."

A 2004 study, published in the journal *Biological Psychiatry,* specifically identified one of the genes responsible for the high incidence of schizophrenia among Blacks in the US. The goal of the study was, in its own words, "to investigate the relationship between the NOTCH4 gene and schizophrenia in African American ("AA") and European American ("EA") subjects."[27]

The scientists studied two SNPs "at the NOTCH4 locus in 123 AA schizophrenia patients, 223 EA schizophrenia patients, 85 AA healthy control subjects, and 211 EA healthy control subjects." The specific markers are called -1725T/G and -25T/C.

The results of the study concluded that the haplotype -1725G/-25T associates to schizophrenia in AA subjects, but not in EA subjects.[28]

"The haplotype -1725G/-25T at the NOTCH4 locus, which results from SNPs of NOTCH4 that are in LD, may increase susceptibility to schizophrenia in AAs. Any effect of this locus on risk for schizophrenia is population-specific."[29]

In the UK, an identical pattern can be found. The BBC reported in 2000 that a study by the UK's Institute of Psychiatry revealed that "African-Caribbean" people are "six times more likely than Whites to be diagnosed as schizophrenic."[30]

An official British government study, named A*esop (Aetiology and Ethnicity in Schizophrenia and other Psychoses,* conducted by psychiatrists and epidemiologists at the social psychiatry unit of the Institute of Psychiatry at the Maudsley hospital in south London—which serves a heavily Black part of the capital city—

27 "NOTCH4 gene haplotype is associated with schizophrenia in African Americans," Xingguang Luo et al., *Biological Psychiatry,* Volume 55, Issue 2, January 15, 2004, Pages 112–117.
28 *Ibid.*
29 *Ibid.*
30 "UK life blamed for ethnic schizophrenia," *BBC,* Friday, July 14, 2000.

found that members of the "African Caribbean community are nine times more likely to suffer from schizophrenia than people in the White community" and described it as an "epidemic."[31]

A 2008 report in the *British Journal of General Practice* also stated that the incidence of schizophrenia in black Caribbeans living in the UK was nine times higher than in the White British population, and, even more disturbingly, that it was even higher in second-generation Black Caribbeans.[32] The report revealed that these Black schizophrenia patients are "more likely to be unemployed, to have a record of convictions, [and] to be resident in decaying inner-city areas."[33]

MAOA: The "Aggression" Gene

The "aggression" gene, also called the "warrior" gene in some circles, called MAOA (monoamine oxidase A, located at Xp11.3, X-Chromosome, short arm, position 11.3) and its variants, is yet another factor that influences crime rates.

Monoamine oxidases (MAOs) are enzymes that are involved in the breakdown of neurotransmitters such as serotonin and dopamine and are, therefore, capable of influencing feelings, mood, and behavior of individuals. If there is a mutation to the gene that is involved in the process of promoting or inhibiting MAO enzymes, it affects a person's personality or behavior and makes them more prone to aggression.[34]

Having the gene does not make that individual automatically a violent criminal, but it has been definitively shown that it is prominently found among such offenders.[35]

31 "Schizophrenia 'epidemic' among African Caribbeans spurs prevention policy change," *The Guardian,* December 9, 2009.

32 "Schizophrenia in black Caribbeans living in the UK: an exploration of underlying causes of the high incidence rate," *British Journal of General Practice,* June 1, 2008; 58(551): 429–434.

33 *Ibid.*

34 "Two genes linked with violent crime," *BBC News,* October 28, 2014.

35 Just three of the first wave of studies on the topic included these significant papers: "The VNTR 2 repeat in MAOA and delinquent behavior in adolescence and young adulthood: associations and MAOA promoter activity," Guo G,

The MAOA gene was first detected in a violent Dutch criminal family in 1993, which served as an explanation why successive generations of that clan were all depraved criminals.[36]

Successive studies honed the topic even further, and, in the words of one of the many studies on the subject:

"This study, drawing on approximately 1,100 males from the National Longitudinal Study of Adolescent Health, demonstrates the importance of genetics, and genetic-environmental interactions, for understanding adolescent delinquency and violence.

"Our analyses show that three genetic polymorphisms—specifically, the 30-bp promoter-region variable number tandem repeat (VNTR) in MAOA, the 40-bp VNTR in DAT1, and the Taq1 polymorphism in DRD2—are significant predictors of serious and violent delinquency when added to a social-control model of delinquency."[37]

The first indicator that the gene was linked to other genetic conditions such as low IQ came in 2007: "High testosterone, maternal tobacco smoking during pregnancy, poor material living standards, dropping out of school, and low IQ predicted violent behavior are associated with men with the low-activity alleles."[38]

Ou XM, Roettger M, Shih JC, *European Journal of Human Genetics.* 16 (5): 626–34, January 23, 2008; "The VNTR 2 repeat in MAOA and delinquent behavior in adolescence and young adulthood: associations and MAOA promoter activity," Guang Guo, Xiao-Ming Ou, Michael Roettger & Jean C Shih, *European Journal of Human Genetics* volume 16, pages 626–634 (2008); and "The integration of genetic propensities into social-control models of delinquency and violence among male youths," Guo G, Roettger M, Shih JC, *American Sociological Review,* 73 (4): 543–568, August 2008.

36 "Does the 'aggressive gene' lurk in a Dutch family?" *New Scientist,* October 30, 1993.

37 "The Integration of Genetic Propensities into Social-Control Models of Delinquency and Violence among Male Youths," Guang Guo, Michael E. Roettger, Tianji Cai, *American Sociological Review,* August 1, 2008.

38 "Early Trauma and Increased Risk for Physical Aggression during Adulthood: The Moderating Role of MAOA Genotype," Giovanni Frazzetto et

Genetics Reveals MAOA Gene More Prevalent in Africans

It was only after the link between the MAOA gene, violence, and low IQ was announced, that researchers began seriously studying the link between race and the gene's prevalence.

Up until then, the prevalence of the MAOA gene in all races had not allowed a clear racial differentiation to be made, but as genetic research techniques were refined, the evidence soon became apparent.

A 2006 study, published in the *Biological Psychiatry* journal, pointed out that "studies with white males have shown that genotypes associated with high levels of monamine oxidase A (MAOA) protect against the impact of childhood maltreatment and adversity on the development of antisocial behavior and conduct disorder," but this "protective effect was not found for non-White abused and neglected individuals."[39]

A lengthy article published in the journal *Nature* in October 2009, dealt with a court case in Italy in which an Algerian immigrant murderer received a lighter sentence because he had the MAOA "aggression" gene and, an appeal court ruled, was therefore not entirely responsible for his actions.[40] There have been at least fifty such instances where this defense has been used in court cases in Europe and America since then, with varying degrees of success.

In 2013, a study published in the journal *Personality and Individual Differences* revealed that the 2-repeat allele MAOA gene was nearly completely absent in whites:

"A line of research has revealed that a polymorphism in the promoter region of the MAOA gene is related to antisocial

al., *PLOS One*, 2 (5): e486, May 30, 2007.

39 "MAOA and the 'cycle of violence:' childhood abuse and neglect, MAOA genotype, and risk for violent and antisocial behavior," Cathy Spatz Widom, Linda M Brzustowicz, *Biological Psychiatry*, Oct. 1, 2006; 60(7):684–9.

40 "Lighter sentence for murderer with 'bad genes': Italian court reduces jail term after tests identify genes linked to violent behaviour," *Nature*, October 30, 2009.

phenotypes. Most of these studies examine the effects of low MAOA activity alleles (2-repeat and 3-repeat alleles) against the effects of high MAOA activity alleles (3.5-repeat, 4-repeat, and sometimes 5-repeat alleles), with research indicating that the low MAOA activity alleles confer an increased risk to antisocial phenotypes.

"The current study examined whether the 2-repeat allele, which has been shown to be functionally different from the 3-repeat allele, was associated with a range of antisocial phenotypes in a sample of males drawn from the National Longitudinal Study of Adolescent Health.

"Analyses revealed that African-American males who carried the 2-repeat allele were, in comparison with other African-American male genotypes, significantly more likely to be arrested and incarcerated.

"Additional analyses revealed that African-American male carriers of the 2-repeat allele scored significantly higher on an antisocial phenotype index and on measures assessing involvement in violent behaviors over the life course. The effects of the 2-repeat allele could not be examined in Caucasian males because only 0.1percent carried it."[41]

In 2014, a paper published in the *Psychiatry Quarterly* journal revealed that "African American males who carry the 2-repeat allele [of the MAOA gene] are significantly more likely than all other genotypes to engage in shooting and stabbing behaviors and to report having multiple shooting and stabbing victims.[42]

That study was at pains to point out that this 2-repeat allele was completely independent of environmental influences.

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41 "Exploring the association between the 2-repeat allele of the MAOA gene promoter polymorphism and psychopathic personality traits, arrests, incarceration, and lifetime antisocial behavior," Beaver KM, et al., *Personality and Individual Differences,* Volume 54, Issue 2, January 2013, Pages 164–168.

42 "The 2-repeat allele of the MAOA gene confers an increased risk for shooting and stabbing behaviors," Kevin M Beaver, J C Barnes, Brian B Boutwell, *Psychiatry Quarterly,* Sept. 2014; 85(3):257–65.

CHAPTER ELEVEN
THE SEVEN MAJOR RACES NAMED

It will be recalled from chapters three and four that genetics clearly divides the world's population into seven distinct categories.

These categories are as follows:

1. Africa (Sub-Saharan).
2. East Asia.
3. Central Asia.
4. Middle East.
5. Oceania.
6. Europe.
7. Americas.

It should be remembered that these are broad categories, and there are individual differences under those headings. Within each major group, there are subdivisions, called "subraces." These subraces diverge slightly in some physical manner, but still share enough genetic commonality with the other members of their major category.

The naming of races has been a long and ongoing matter of debate since the first scientific writings on the topic. There can be little doubt that many of the earlier categorizations were based on opinions rather than any conclusive genetic facts.

The advent of genetics has changed this completely, and it is now possible to give these categories names which reflect the indigenous racial makeup of each region. In choosing these names, it is sensible to use, as far as possible, terms which are easily un-

derstood and commonplace, rather than inventing new terms which generally only serve to confuse.

Using this understanding as a basis, we can therefore give names to the racial categories as they have been identified by DNA:

1. Region: Europe. Racial group name: "Europid."

The name "Europe" comes from Greek mythology, and therefore dates to at least 800 BC. "Europid" is an all-encompassing term for the light-skinned people of Europe, who have essentially three major subgroups:

1.1 Nordics, with fair hair, fair eyes, "long"[1] skulls, and tall stature.

1.2 Alpines, with brunet hair, light brown eyes, "square" skulls, and shorter stature, and

1..3 Mediterraneans, or "Old Europeans," with dark hair, dark eyes, and "square" skulls.

Other subtypes also exist, but these often consist of a mixture of these three main subgroups or have some other outside or ancient influence.

2. Region: Africa (sub-Saharan). Racial group name: "Negroid."

The peoples of this area can be broadly called by the well-understood term "negroid."[2]

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1 A brief discussion on skull types, and the scientific names used, is contained in the next chapter.

2 The word "negro" is derived from the Latin word which means "black" ("niger"). It has no further connotations than that, and this is borne out by the fact that at least two present-day African states—one of which is the largest country on the African continent—Niger and Nigeria, appear to derive their names from this word. The name was first given to that part of the world by the famous Christianized Moorish geographer, Leo Africanus (born al-Hasan circa 1494, died circa 1554) in his 1550 work *Della descrittione dell'Africa et delle cose notabili che ivi sono*. In that book, the first accurate geography of Africa, Africanus named a large river in West Africa as the "Niger River," a name it bears to the present day. Niger and Nigeria have taken their names from this river. It should be said that some scholars have since claimed that that

Two major negroid subraces can be identified according to their genetic groupings:

2.1 The *Bantu,* who make up the largest single grouping, and whose appearance most typically represents what is popularly regarded as "racially African." This is characterized by dark brown to black skin, short peppercorn hair, broad noses, dark brown to black eyes, and very low cephalic indexes.

The word "Bantu" is derived from the southern African word for "people" (*abantu*) as used in both the Zulu and Xhosa languages. The Pygmies of West and Central Africa, long regarded as a separate race, only diverge from the Bantu in terms of some growth-related genes.

2.2 The Khoi-San, and other pre-Bantu peoples. Today mainly found in southern Africa and previously known as "Bushmen." They are characterized by a near epicanthic fold in the eyelid form, and a lighter brown skin.

Other groups within sub-Saharan Africa commonly called "African" (such as the Sudanese, and other northeast peoples) are in fact the product of racial admixture from the Middle East.[3]

3. Region: East Asia. Racial group name: "Mongoloid."

The East Asian people make up those indigenous races of China, Taiwan, Japan, Mongolia, and Korea. As this area includes a large number of languages and subraces, it makes sense to use the traditional term of "Mongoloid" to define them.

The major subraces within the Mongoloids are:

3.1 North Mongoloid: Ethnic groups include the Altai and Siberians. Characterized by pale skin and prominent epicanthic folds on their small oval-shaped eyes.

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river name was based on an Arabic or Berber word, but there is no conclusive evidence for this.

3 The existence and geographic spread of mixed races can be seen in several regions of the world. Some of the prime examples include almost all North Africa, Turkey, Central and South America. This is not to say that all individuals from those regions are of mixed origin, but a considerable number—certainly the majority—are.

2.2 Central Mongoloid: Ethnic groups include the Sino-Tibetan (the largest group of which are the Han Chinese and the Japanese). Characterized by "yellowish" fair skin, inner double eyelid (slight epicanthic fold), and medium-sized, oval shaped eyes.

2.3 Southern Mongoloid: Ethnic groups include Cambodians, Malays, Indonesians, Javanese, and Filipinos). Characterized by varying degrees of skin color from light to dark brown, with outer double eyelid (no epicanthic fold) and larger rounder eyes. These physical characteristics are likely to be derived from admixture with West Mongoloids (see below).

All these groups have straight, jet black hair.

4. Region: Central Asia. Racial group name: "Indo-Iranoid."

When the East Asian genetic component of Central Asia is excluded, the region includes all the territories from the western borders of China to the Caspian Sea. It includes the Indian subcontinent, Afghanistan, Iran, and the former Soviet republics of Kazakhstan, Kyrgyzstan, Tajikistan, Turkmenistan, and Uzbekistan.

This is a wide area, but genetically speaking much of the region can be directly connected to the spread of the Indo-European language, hence it is logical to give them the name "Indo-Iranoid."

On the Indian subcontinent, the Indo-Iranoids are divided into two groups, split approximately on geographic lines. The north is characterized by lighter skins, with dark to black eyes and dark hair (and very occasional flashes of Europid ancestry). The south is characterized by much darker skins, and in some areas, even negroid-approaching physiognomies. These physical differences form the basis of India's famous caste, or class system.

To the north and northwest of India, the Indo-Iranoids are generally of lighter complexion, and although black eyes are predominant, lighter hair and eyes are present in numerically significant numbers, an indication of ancient European admixture. This includes the Turkic peoples.

5. Region: Middle East. Racial group name: "Semitic."

These are the people who originate from the Saudi Arabian Peninsula and its immediate surrounding areas. They are the people broadly termed "Arabs," although there is much confusion over who exactly is an "Arab."

It is incorrect to label the entire Middle East as "Arab," or the Muslim part of North Africa as "Arab," as those regions are now largely of mixed-racial descent.

It is therefore convenient to name the indigenous inhabitants of the Middle East after their language, "Semitic," rather than their region, as this more closely defines who they are. The use of this term limits the definition of Middle Easterners to those who share common genetics.

The two most important groups of Semites are the Saudis and the Jews, both of whom share very similar DNA. Both of these groups are characterized by light brown skin color, black eyes, black hair, and prominent noses, which may or may not be the result of ancient admixture with outside groups.

Jews are in turn divided into two main groups: Ashkenazim and Sephardic. The main difference between the two groups is that the Ashkenazim have on average 30 percent Europid admixture, resulting from their extended residence in Europe. This is the cause of the lighter complexions, lighter eyes, and lighter hair color among a significant number of Ashkenazim Jews.

6. Region: Oceania. Racial group name: "Australoid."

The Oceania region includes the island nations north of Australia, Australia itself, and the South Pacific Islands. This includes the indigenous peoples of the islands to the immediately north of Australia.

For this reason, it is convenient to label this group "Australoid," after the most prominent of these groups, as it is a term which immediately conjures up both a specific geographical location and a broadly common phenotype.

There are two main ethnic groups in this region:

6.1 Australian Aborigines. Characterized by brown to dark skin, dark eyes, and curly (but *not* peppercorn) hair.

6.2 Papuans and related tribes: Sharing much genetic material with the Aborigines of Australia, these tribes are however characterized by much darker skins and near-negroid physical traits.

7. Region: Americas: Racial group name: "Amerindians."

Although originally from East Asia, these people have been isolated in the Americas for so long that the process of genetic drift[4] has allowed them to form a genetic group on their own.

The "purer" forms appear in the present day more toward Central and South America, and are characterized by a shorter stature, more "Asiatic" facial appearance, darker skins, and black eyes. The large scale importation of negroid types during the Trans-Atlantic slave trade has greatly affected their phenotype in many regions, and this, along with a Spanish colonialist input, has created what many call the "Mexican" or "Hispanic" type (although there is no such race as "Hispanic" in any genetic sense.

In North America, the "Indians" are characterized by much lighter skins, and less "Asiatic" faces—all mostly the result of European admixture.

It is therefore possible to use the terms Europid, Negroid, Semitic, Mongoloid, Indo-Iranoid, Australoid, and Amerindian to properly categorize the seven major races as defined by DNA.

4 See Chapter Two.

CHAPTER TWELVE
THE SEVEN MAJOR RACES — A TYPOLOGICAL OVERVIEW

Having established that genetics has clearly defined seven major races within mankind, it is valuable to undertake a typological overview of these major races, and their subraces.

The reader should always consider that all physical differences are the result of genetics. Genes, or rather specific areas within genes (SNPs), determine all aspects of a human's physical appearance, and thus of that individual's racial appearance.

The Cephalic Index as a Measure of Race

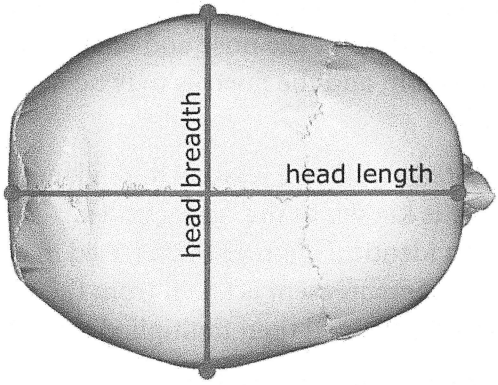
Even a casual observer will have noticed that some people have what appear to be "long" heads, while others appear to have "round" faces. This appearance is of course genetic in origin.

Anthropologists have long been aware of the difference in skull shape and have created what is known as the "Cephalic Index" (or CI) in order to identify certain shapes.

The CI is a number which is generated by a simple equation: it is the breadth of the head divided by the length of the head, and then expressed as a percentage by multiplying by 100.

Written out, the equation looks like this:

$$\text{Cephalic Index} = \frac{\text{head breath}}{\text{head length}} \times 100$$

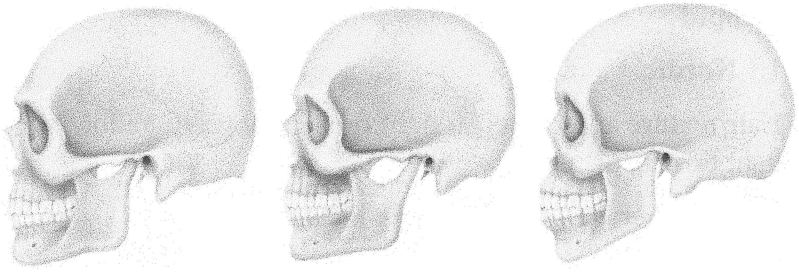


The Cephalic Index as shown by measurements on a skull.

When the CI ratio falls below 75, the skull is termed "dolichocephalic," or "narrow-headed." When the CI ratio exceeds 80, the skull is termed "brachycephalic," or broad-headed. When the CI ratio falls between 75 and 80, the skull is termed "mesocephalic," or "medium-headed." This is usually the result of admixture between dolichocephalic and brachycephalic individuals.

Facial Features as a Measure of Race

When the lower part of the face, most notably in the jaw line, projects out beyond the rest of the facial line, it is called *prognathous* (or *prognathic*). A non-prognathic jawline is called *orthognathous* (or *orthognathic*).



From left to right: A (northern European) skull, an Amerind skull, and a sub-Saharan African skull. The illustration highlights the prognathic nature of sub-Saharan Africans. "Africans" who do not display this prominent feature are of admixed racial ancestry.

The nose alae, the so-called "wings" of the nose, located on both sides on the bottom of the nose and enclosing the nasal inlets, vary considerably, and the nostrils may be narrow, medium, broad, or round.

When calculated as a figure relative to the length of the nose, the result is what is known as the "nasal index." The nasal index is calculated in an identical manner as that used in generating the cephalic index. A measurement is taken from the root of the alae to the angle with the lip, and then multiplied by 100.

The genetic basis of the nasal index serves as a highly accurate indicator of racial ancestry, particularly in populations with large mixed-race ancestries such as India or northeast Africa.

A long and narrow nose is known as *leptorrhine* and has a nasal index of between 55 and 69. A moderately shaped nose is known as *mesorrhine* and has a nasal index of between 70 and 84. A broad and short nose is known as *platyrrhine* and has a nasal index of between 85 and 99. Nasal indexes below 55 are called *hyperleptorrhine*, and those above 100 are known as *hyperplatyrrhine*.

RACIAL TYPES

1. Europid

1.1 Nordic

Hair texture and color: Straight or slightly wavy. Yellow, very light brown, or red.

Stature: Average 1.73 meters (68 inches).

Skin color: Very fair.

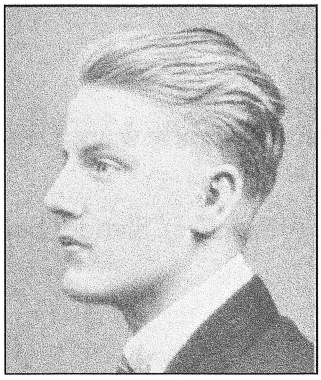
Eye color: Light (blue, gray, occasionally green).

Cephalic Index: 76–79. Dolichocephalic.

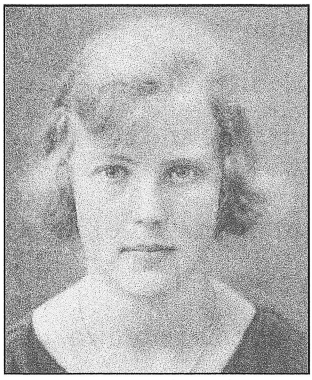
Nose: *Leptorrhine,* straight or aquiline nose. Narrow face.

Average IQ Level: 105

Location: Scandinavia, Northwestern Europe, Baltics, White Russia.



Nordic male.



Nordic female.

1.2 Alpine

Hair texture and color: Straight, or slightly wavy. Brunette, brown.

Stature: 1.635 meters (64 inches). Usually thick-set body.

Skin color: Fair to medium tawny.

Eye color: Brown.

Alpine male.

Alpine female.

Cephalic Index: 82–86; brachycephalic.

Nose: *Mesorrhine,* straight, rarely aquiline nose.

Average IQ Level: 99.5

Location: Central and Southeast Europe.

1.3 Mediterranean

Hair texture and color: Straight or slightly wavy. Dark brown or near black.

Stature: Average 1.615 meters (63 inches). Slender build.

Skin color: Fair.

Eye color: Dark brown.

Cephalic Index: 72–75; dolichocephalic. Face narrow, oval.

Nose: *Leptorrhine,* generally straight.

Average IQ Level: 97

Location: Southern Mediterranean coast, Spain, Ireland, Wales, France, United Kingdom.

Note: there are numerous other Europid subraces, depending upon the anthropologist consulted. For brevity's sake, just the three above are listed, as it would appear that many of the others are for the greatest part either slight differentiations on the above three, or combinations thereof.

Mediterranean male.

Mediterranean female.

2. Negroid

2.1 Bantu

Hair texture and color: Hard, peppercorn. Black.

Stature: Average 1.68 meters (66 inches).

Skin color: Dark brown to pitch black.

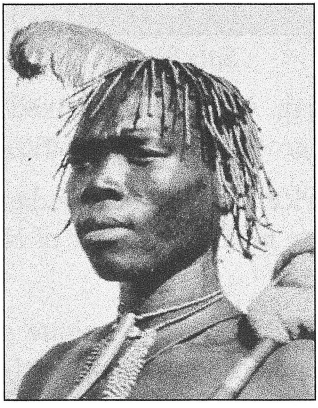
Eye color: Dark brown to pitch black.

Cephalic Index: 75–80; Mesocephalic, with tendency toward brachycephalic. Prognathicism very common, sometimes marked.

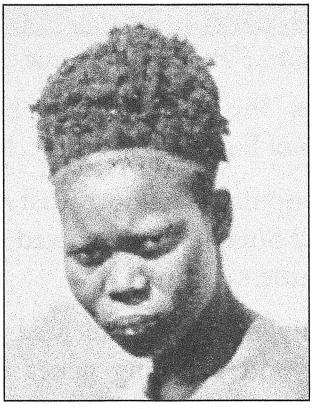
Nose: *Platyrrhine,* broad and flat, often wide nostrils.

Average IQ Level: 67

Location: Central to southern Africa.



Bantu male.



Bantu female.

2.2 Khoi-San

Hair texture and color: Hard, peppercorn, sparse. Black.

Stature: Average 1.5 meters (59 inches).

Skin color: Brown to light brown.

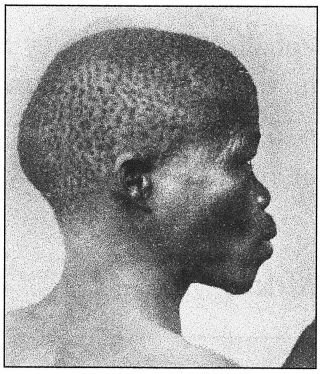
Eye color: Dark brown to pitch black.

Cephalic Index: 75–77; Mesocephalic, with tendency toward brachycephalic. Prognathicism very common, sometimes marked.

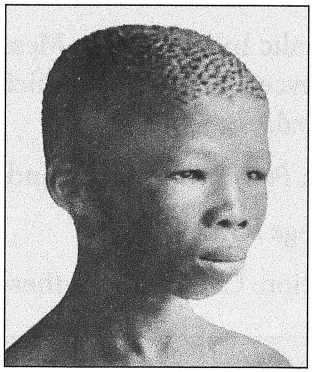
Nose: *Platyrrhine,* broad and flat, often wide nostrils.

Average IQ Level: 55

Location: Southern Africa.



San ("Bushman") male.



San female.

Note: The inhabitants of North Africa are a mixed group made up of European, Semitic, Negroid, and even Asiatic strains.

The population of northeast Africa, including the Sudan, Ethiopia, and Somalia, are a mixed race made up mainly of Negroid and Semitic mixture.

The Pygmies and the Maasai peoples have unique growth stunting and inducing genes respectively, otherwise they are identical to the Bantu. The tallest tribe in Africa, the Dinka from the Sudan, are of mixed Semitic-Negroid ancestry.

3. Semitic

Hair texture and color: Straight. Black.

Stature: Average 1.66 meters (65 inches).

Skin color: Brown to light brown.

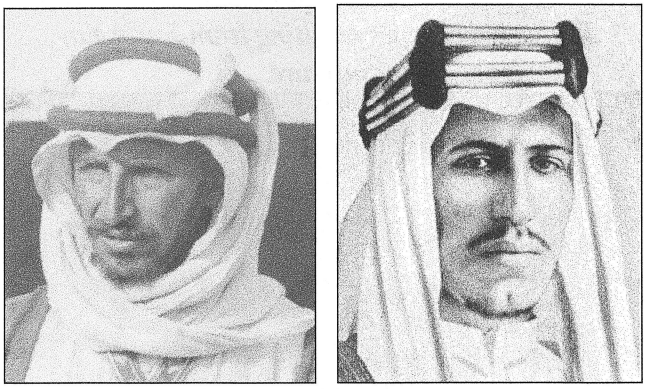
Eye color: Dark brown to black.

Cephalic Index: 75–79; Mesocephalic, with tendency toward dolichocephalic.

Nose: *Leptorrhine,* straight or aquiline nose. Thin face, angular features. Many have a pronounced nose, with this feature having become prominent among most Jews.

Average IQ Level: 79

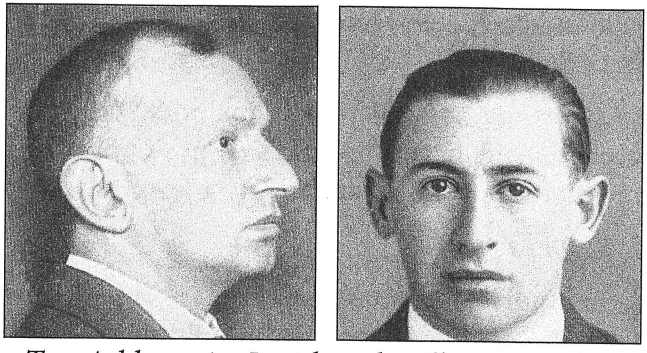
Location: Saudi Arabia, now spread out into many parts of the Middle East. Jews have spread out into European nations.



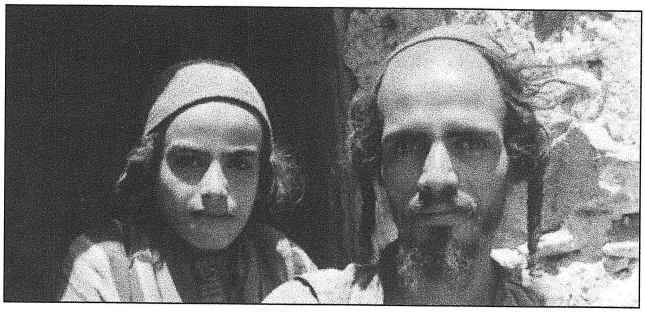
Two Semitic males from Saudi Arabia. Right, a former Saudi Emir.

3.1 Jews.

The Jewish subrace is differentiated from the pure Semites only by the degree of non-Semitic admixture, either from Europids or Indo-Iranoids. Ashkenazim (European Jews) have the largest Europid ancestry, and therefore display the most Europid features. On average, Ashkenazim Jews have around 30 percent Europid ancestry, and some have far more. This causes their phenotype to vary greatly, and they can display a range from fair hair, eyes, and skin color, to very dark features. Hair texture also varies widely, ranging from straight to very curly. Sephardic Jews retain the greatest genetic similarity to the Semitic race, and on average have around 10 percent non-Semitic ancestry. This causes them, on average, to be darker in all respects than Ashkenazim Jews. The one



Two Ashkenazim Jewish males, illustrating their diverse phenotypes resulting from European admixture.



Sephardic Jews from Iraq.

common and striking attribute appears to be the famous "Jewish nose" which is a dominant trait in both Ashkenazim and Sephardic Jews.

Average IQ level of Ashkenazim Jews: 100

Average IQ level of Sephardic Jews: 84

4. Indo-Iranoids

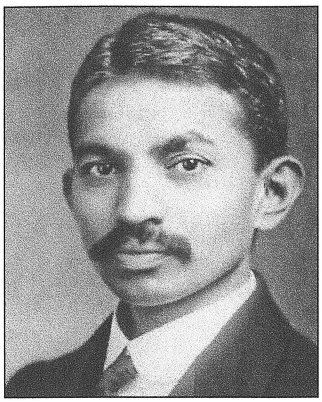
4.1 India:

4.1.1 Dravidian (India)

Hair texture and color: Straight. Black.

Stature: Average 1.63 meters (64 inches).

Skin color: brown to black skin, light skin in those with large Europid ancestry.



Dravidian male.

Dravidian female.

Eye color: Black, light eyes in those with Europid ancestry.

Cephalic Index: 73–76; generally dolichocephalic.

Nose: *Mesorrhine,* tendency to *leptorrhine.*

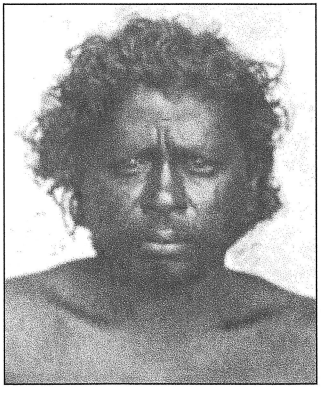
Average IQ Level: 87

Location: Indian subcontinent, north India, Punjab, Pakistan. Associated with later settlers of the region.

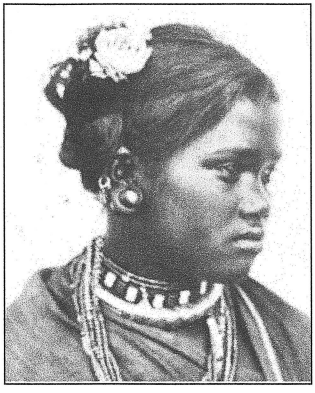
4.1.2 Pre-Dravidian

Hair texture and color: Straight, tendency to curly in some regions. Black.

Stature: Average 1.5 meters (59 inches).



Pre-Dravidian male.



Pre-Dravidian female.

Skin color: Brown to black.

Eye color: Black.

Cephalic Index: 78, mesocephalic.

Nose: *Mesorrhine,* tending to *platyrrhine.*

Average IQ Level: 72

Location: South India, Sri Lanka, parts of Malay, and bordering island regions.

4.2 Iranoid

Hair texture and color: Straight, slightly curly. Black to brown, very occasionally fair (Europid ancestry).

Stature: Average 1.69 meters (66 inches).

Skin color: White to light brown.

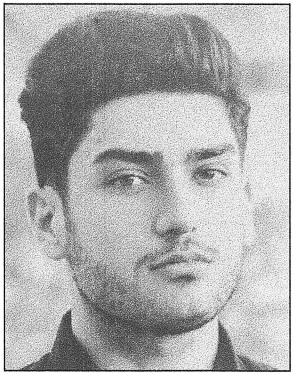
Eye color: Brown, occasionally blue depending on amount of Europid ancestry.

Cephalic Index: 85, brachycephalic, tendency to dolichocephalic.

Nose: *Leptorrhine.*

Average IQ Level: 87

Location: Central Asia, Iran, Iraq, Afghanistan, Turkmeni-



Iranoid male.

Iranoid females

stan, Uzbekistan, Tajikistan, Kazakhstan, Azerbaijan, and related regions.

5. Mongoloid

5.1 Han Chinese

Hair texture and color: Straight. Black.

Stature: Average 1.65 meters (64 inches).

Skin color: Light.

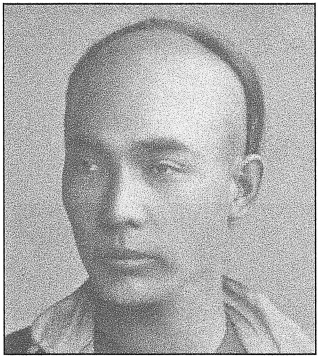
Eye color: Dark brown, notable for epicanthic fold. Eye shape tends to angular.

Cephalic Index: 76 – 80, mesocephalic, strong tendency to a high forehead. Broad face

Nose: *Platyrrhine,* tendency to *mesorrhine.*

Average IQ Level: 105

Location: Mainland China, Taiwan. The single largest group of the Mongoloid race.



Han male.

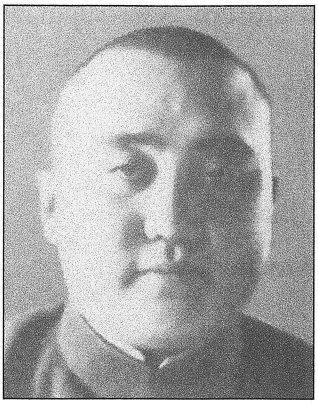
Han female.

5.2 Mongols

Hair texture and color: Coarse, straight. Black.

Stature: Average 1.65 meters (64 inches).

Skin color: Yellowish-brown.



Mongol male.

Mongol female.

Eye color: Dark brown. Eye shape "straighter" than Han Chinese.

Cephalic Index: 84–87; Brachycephalic.

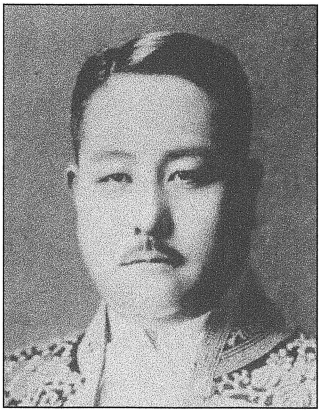
Nose: Strong tendency to *platyrrhine.*

Average IQ Level: 100

Location: Northwest of China, Mongolia, region around Lake Baikal on southern extremities of Russia.

5.3 Yayoi (Japan)

Hair texture and color: Straight. Black.



Yayoi male.

Yayoi female.

Stature: Average 1.71 meters (67 inches).

Skin color: Light.

Eye color: Dark brown to black. Eye shape tends to round or oval.

Cephalic Index:79–81; Mesocephalic, tendency toward brachycephalic.

Nose: *Mesorrhine,* tendency toward *platyrrhine.*

Average IQ Level: 105

Location: Japan (South), parts of Korea, east coast of China. Northern Japan is home to a group known as the Ainu, who tend toward more Europid features, and have notably more body and facial hair.

5.4 Far North Asiatic

Hair texture and color: Straight. Black.

Stature: Average 1.51 meters (59 inches).

Skin color: Light brown.

Eye color: Dark brown.

Cephalic Index: 78–80; Brachycephalic.

Nose: *Mesorrhine,* tendency to *platyrrhine.*

Average IQ Level: 91

Siberian Arctic couple.

Location: Artic Circle from northern Scandinavia to Siberia and the far east of Russia.

6. Australoid

6.1 Negrito

Hair texture and color: Very curly, tending to peppercorn. Black.

Stature: Average 1.49 meters (58 inches).

Skin color: Black.

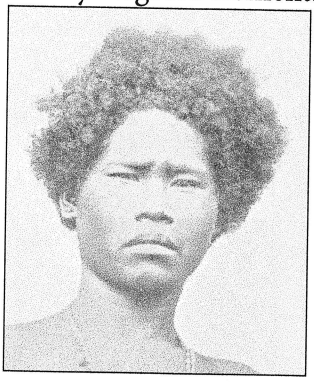
Eye color: Black.

Cephalic Index: 73–75; dolichocephalic, tendency to brachycephalic.

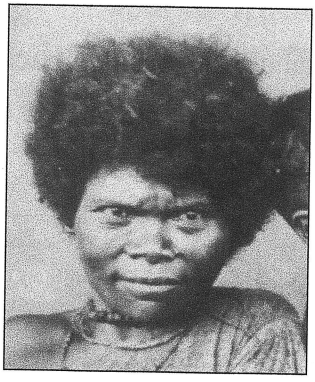
Nose: *Platyrrhine.*

Average IQ Level: 68

Location: South East Asian island regions, Malay Peninsula, Philippines, New Guinea, Melanesia, Admiralty Islands to New Caledonia; Fiji and East Sumatra. Appears to be related to an early Negroid element.



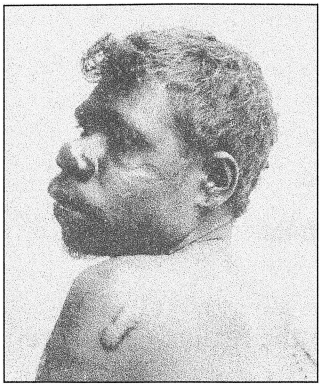
Negrito male.



Negrito female.

6.2 Aborigine

Hair texture and color: Curly, sometimes wavy or even straight. Brown or black.



Aborigine male.

Aborigine female and child.

Stature: Average 1.66 meters (65 inches).

Skin color: Brown to dark brown.

Eye color: Dark brown.

Cephalic Index: 75; dolichocephalic. Prognathic, prominent brows.

Nose: *Platyrrhine.*

Average IQ Level: 62

Location: Australia and surrounding islands.

6.3 Bornean

Hair texture and color: Straight. Black.

Borneo female and male.

Stature: Average meters (inches).

Skin color: Brown.

Eye color: Dark brown.

Cephalic Index: 82–84; Brachycephalic.

Nose: *Mesorrhine,* tendency to *platyrrhine.*

Average IQ Level: 83

Location: Borneo and other islands in the region. Closely related to New Zealand Maoris.

6.4 Maori

Hair texture and color: Straight. Black.

Stature: Average 1.7 meters (66 inches).

Skin color: Light brown to dark brown.

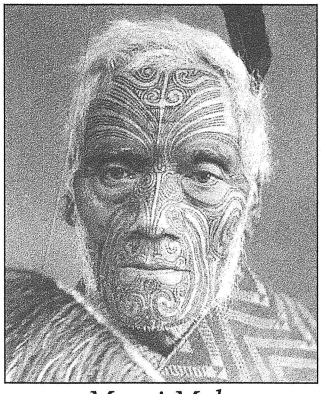
Eye color: Dark brown.

Cephalic Index: 73–75; Dolichocephalic.

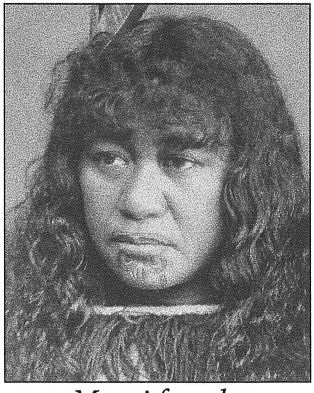
Nose: *Mesorrhine.*

Average IQ Level: 90

Location: New Zealand. Genetically, the origins of the Maori are in East Asia, but they have heavily admixed with other South East Asians during their ancient journey south.



Maori Male.



Maori female.

6.5 Polynesian

Hair texture and color: Straight. Black.

Stature: Average 1.72 meters (68 inches).

Skin color: Light brown.

Eye color: Dark brown.

Cephalic Index: 79–81; Mesocephalic, with tendency toward brachycephalic.

Nose: *Mesorrhine.*

Average IQ Level: 90

Location: South Pacific Islands.

Polynesia male.

Polynesia female.

7. Amerindian

7.1 Far North Amerind (Inuit, or "Eskimo")

Hair texture and color: Straight. Black.

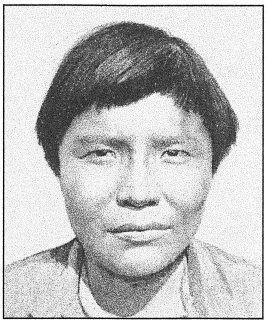
Skin color: Yellow-Brown.

Eye color: Black. Eyelids occasionally with epicanthic fold.

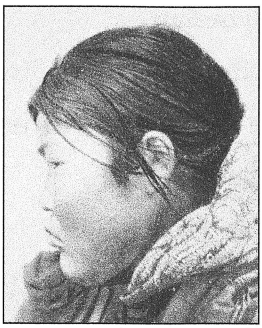
Stature: 1.58 meters (62 inches).

Cephalic Index: 72. Dolichocephalic head with flat face.

Nose: *Platyrrhine.*



Far North Amerind, Inuit male.



Far North Amerind, Inuit female.

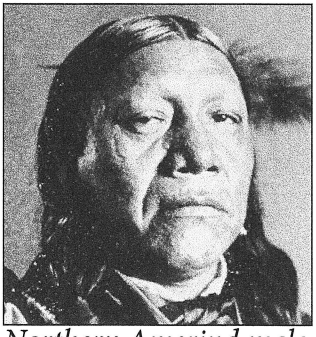
Average IQ Level: 91

Location: North America, Greenland, and the extreme northeast of Asia. Closely related to the "Far North Asiatic."

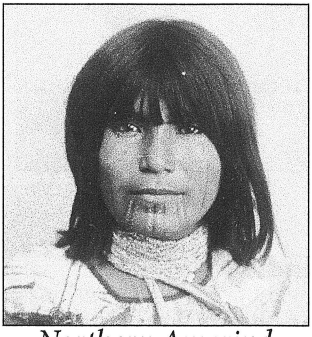
7.2 Northern Amerind

Hair texture and color: Straight. Black.

Stature: Average 1.70 meters (67.5 inches).



Northern Amerind male, Pawnee Indian.



Northern Amerind, Apache Indian.

Skin color: Light brown.

Eye color: Dark brown.

Cephalic Index: 73–78; dolichocephalic to mesocephalic.

Nose: *Leptorrhine,* straight, or aquiline nose. Large oval face.

Average IQ Level: 85

Location: Plains and Northern and Eastern Woodlands.

7.3 Central Amerind

Hair texture and color: Straight. Black.

Skin color: Light brown.

Eye color: Dark brown.

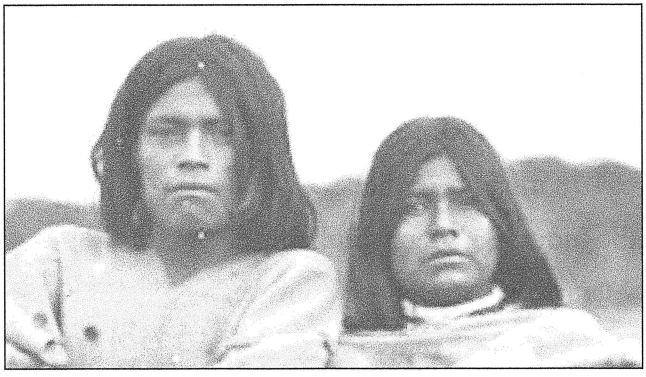
Stature: Average 1.67 meters (65 inches).

Cephalic Index: 80–89; brachycephalic.

Nose: Mesorrhine, straight or concave, rarely aquiline.

Average IQ Level: 83

Location: Central America, South America.



Central Amerind couple, Mexico.

7.4 Southern Amerind

Hair texture and color: Straight. Black.

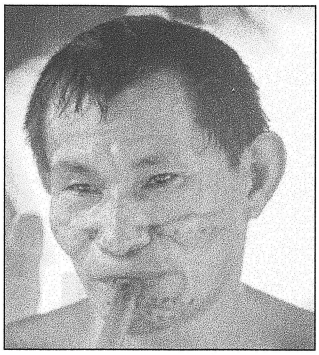
Skin color: Brown.

Eye color: Dark brown to black.

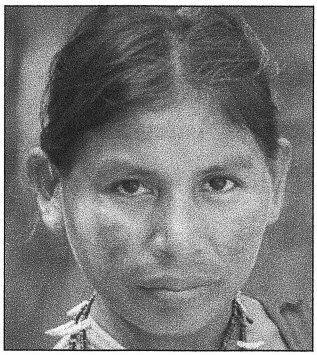
Stature: 1.56 meters (61 inches).

Cephalic Index: 80–89; brachycephalic.

Nose: *Mesorrhine,* straight or concave, rarely aquiline.



Southern Amerind male (Amazon).



Southern Amerind female (Amazon).

Average IQ Level: 78

Location: Central America, South America.

7.5 Far South Amerind

Hair texture and color: Straight. Black.

Skin color: Brown.

Eye color: Dark brown.

Stature: 1.78 meters (70 inches).

Cephalic Index: 85; brachycephalic.

Nose: *Mesorrhine,* straight or concave.

Average IQ Level: 78

Location: Patagonia, Argentina.

Far South Amerind male.

Far South Amerind female.

CHAPTER THIRTEEN:
FIVE COMMON RACIAL MYTHS DEBUNKED

As the reader who has persevered to this chapter will now have realized, the genetic evidence definitively shows the existence of different races, and the fact that race has a biological basis.

This conclusion is also directly contradictory to the opinion that race does not exist and is only a "social construct." It is therefore valuable to deal with a handful of the most commonly spread racial myths which lie at the heart of race-denial.

1. "All humans are 99 percent genetically the same, so there is no genetic difference."

The claim that all humans are "99 percent genetically the same," despite being repeated ad infinitum, was made in 1972—over 30 years before advanced genetic studies and DNA analysis became available. That alone should disqualify the claim of any relevance, given the vast advances in the science in the more than 50 years.

The claim was made by the American biologist Richard Lewontin (1929–2021) in his 1972 paper titled "The Apportionment of Human Diversity."

Using the still crude analytical techniques of his era, and relying on blood protein sampling, Lewontin announced that the number of differences were of "virtually no genetic or taxonomic significance" and that there was no justification of the continuance of racial classifications.

This claim, as has now been conclusively proven, is false, and this fact is widely known in the scientific community—although it is still repeated ad nauseum by the mass media and uniformed commentators.

In fact, as outlined in Chapter One of this book, DNA sequencing was only discovered five years ***after*** Lewontin made his still widely repeated claim that there are no genetic differences between the races. In 2003, a study appeared in the *Bioessays* journal[1] which thoroughly and scientifically disproved Lewontin's assertion.

This article—and others like it—was republished in many other reputable journals, including that of the US Government's National Library of Medicine (which falls under the National Institutes of Health, or NIH, part of the US Government's Department of Health and Human Services).

In that paper, titled "Human genetic diversity: Lewontin's fallacy," the author—a scientist at Cambridge University, UK—exposed Lewontin's 1972 conclusion as "unwarranted because the argument ignores the fact that most of the information that distinguishes populations is hidden in the correlation structure of the data and not simply in the variation of the individual factors."

Lewontin's argument came down to the assumption that race could be determined by simply looking at one trait, and one trait alone.

A theoretical example explains it better: If, say, 60 percent of "Africans" have hazel eyes, and 60 percent of "Europeans" have hazel eyes, then any attempt to predict race based on eye color alone might be incorrect because the prediction would be wrong 40 percent of the time. Therefore, Leowintin concluded, race "does not exist."

The flaw in this "argument" is immediately apparent. As detailed earlier in this book (and by extension, all the scientific

1 "Human genetic diversity Lewontin's fallacy," Edwards, A. F., *Bioessays*, 2003 Aug; 25(8):798–801.

work on genetics of the previous three decades), race is determined by hundreds of different characteristics and genes, all of which had an overall effect of creating the racial categories.

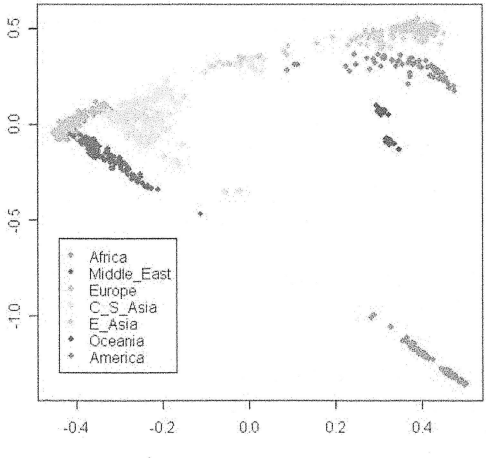
Put another way: Lewontin's "argument" *might,* and the emphasis is on "might," hold true if a single loci is considered. But if a few hundred, or thousands of loci are considered, the clustering along racial lines becomes clear and scientifically irrefutable.

It is not the degree of genetic similarity which "disproves" race, but rather the degree of difference which *does* prove that race matters.

2. "Race has no basis in biology."

The simple answer to this is that the existence (and accuracy) of commercially available home DNA testing kits has definitively proven that race has a biological basis. These home DNA testing kits would not work if there was no biological basis to their scientific methodology.

DNA testing kits accurately predict an individual's inheritance based purely on a saliva sample using the process known as "principal component analysis" (PCA). The DNA testing companies then compare the analyzed results of that sample to their extensive databases, and by observing the clustering pattern of the genetic components, accurately predict individual racial ancestry. This process is well-illustrated by the table (left) from a paper published in the journal *Science* which mapped out genome-wide patterns of variation taken from DNA samples:[2]



2 "Worldwide Human Relationships Inferred from Genome-Wide Patterns of Variation," Jun. Z., et al., *Science,* Feb. 22, 2008, Vol 319, 5866,

3. "Race is a social construct."

This is an increasingly commonly used statement" which claims that the idea of race was only invented by a group of white, Western, capitalists to exploit other people by artificially dividing them into competing groups.

Once again, the simple answer is that the science of genetics has definitively proven that racial groupings match genetic profiles perfectly, and that this scientific reality has nothing to do with an imaginary conspiracy of white capitalists.

Apart from this fact being made obvious by the existence and accuracy of commercially available home DNA testing kits, it has also been admitted to by none other than the Stanford School of Medicine, which in 2005 reported that "racial groupings match genetic profiles."[3]

The report said that "checking a box next to a racial/ethnic category . . . also says something about a person's genetic background."[4]

According to the report, which quoted a study published in the February 2005 issue of the *American Journal of Human Genetics*,[5] which analyzed the responses of 3,636 people who all identified themselves as either white, African-American, East Asian, or Hispanic. Of these, only five individuals had DNA that matched an ethnic group different than the box they checked at the beginning of the study. That's an error rate of 0.14 percent," the Stanford report said.

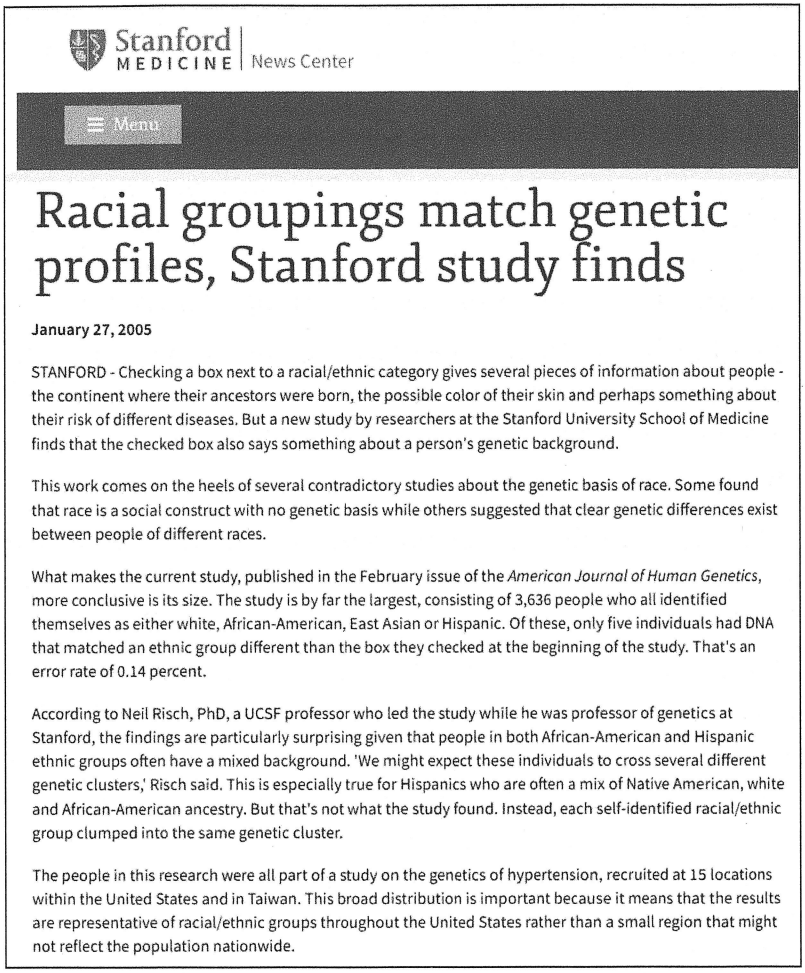
"According to Neil Risch, PhD, a UCSF professor who led the study while he was professor of genetics at Stanford, the findings are particularly surprising given that people in both Afri-

pp. 1100–1104.

3 "Racial groupings match genetic profiles, Stanford study finds," *Stanford Medicine News Center,* January 27, 2005.

4 *Ibid.*

5 "Genetic Structure, Self-Identified Race/Ethnicity, and Confounding in Case-Control Association Studies," Tang, H., et al., *American Journal of Human Genetics,* 76.2 (2205) 268–275.



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Menu

Racial groupings match genetic profiles, Stanford study finds

January 27, 2005

STANFORD - Checking a box next to a racial/ethnic category gives several pieces of information about people - the continent where their ancestors were born, the possible color of their skin and perhaps something about their risk of different diseases. But a new study by researchers at the Stanford University School of Medicine finds that the checked box also says something about a person's genetic background.

This work comes on the heels of several contradictory studies about the genetic basis of race. Some found that race is a social construct with no genetic basis while others suggested that clear genetic differences exist between people of different races.

What makes the current study, published in the February issue of the *American Journal of Human Genetics*, more conclusive is its size. The study is by far the largest, consisting of 3,636 people who all identified themselves as either white, African-American, East Asian or Hispanic. Of these, only five individuals had DNA that matched an ethnic group different than the box they checked at the beginning of the study. That's an error rate of 0.14 percent.

According to Neil Risch, PhD, a UCSF professor who led the study while he was professor of genetics at Stanford, the findings are particularly surprising given that people in both African-American and Hispanic ethnic groups often have a mixed background. 'We might expect these individuals to cross several different genetic clusters,' Risch said. This is especially true for Hispanics who are often a mix of Native American, white and African-American ancestry. But that's not what the study found. Instead, each self-identified racial/ethnic group clumped into the same genetic cluster.

The people in this research were all part of a study on the genetics of hypertension, recruited at 15 locations within the United States and in Taiwan. This broad distribution is important because it means that the results are representative of racial/ethnic groups throughout the United States rather than a small region that might not reflect the population nationwide.

can-American and Hispanic ethnic groups often have a mixed background.

"'We might expect these individuals to cross several different genetic clusters,' Risch said. 'This is especially true for Hispanics who are often a mix of Native American, white, and African-American ancestry. But that's not what the study found. Instead, each self-identified racial/ethnic group clumped into the same genetic cluster.'[6] "'This shows that people's self-identified race/ethnicity is a nearly perfect indicator of their genetic background,' Risch said.

6 *Ibid.*

"Furthermore, when the research team analyzed each of the four clusters, they found two distinct sub-groups within the East Asian genetic cluster—and these two groups correlated with people who identified themselves as Chinese and Japanese.

"None of the other genetic groups could be broken down into smaller sub-sections. This suggests that there isn't enough genetic difference to distinguish between people who have ancestry from northern Europe versus southern Europe, for example."[7]

In other words, DNA testing has definitively shown that the genetic gap between races is far larger than within races, which in turn disproves the "social construct" delusion.

4: "Race is only skin deep."

The answer to this commonly-heard nonsense is so obvious that, once again, it is incredible that anyone even says it, much less believes it.

If race was only a matter of "skin color," then the person saying such a thing must be blind to ignore the many other physical characteristics which define races.

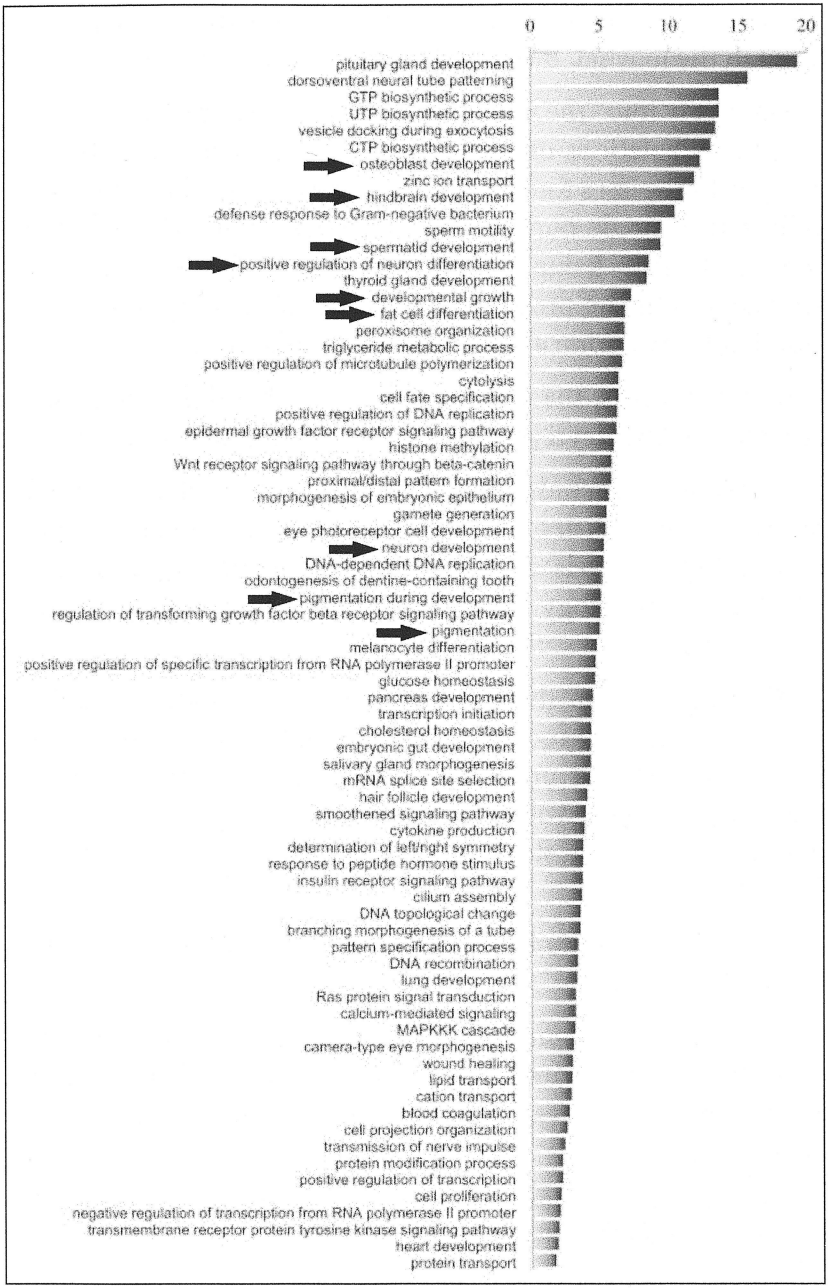
For example, anyone looking at a Chinese person will not say that that person is an "African," nor will they say that an African is "Chinese." Yet these same people will say that race is "only a matter of skin color."

Next page: A chart from the *2011 BMC Evolutionary Biology* paper which details the effects of SNPs upon *human biological processes in different races.*

Some important processes—all controlled by individual genes, are highlighted in that chart..

The highlighted genes control pigmentation, neuron (in the brain) development, fat cell differentiation, positive regulation of neuron differentiation, hindbrain development, and osteoblast development.

7 *Ibid.*



Source: "Different Level of Population Development among Human Genes," Wu, D.D., et al., *BMC Evolutionary Biology*, 11 (2011):16.

The genetic differences between races have been outlined in this book, but it is of value here to quote one more study, published in the journal, *BMC Evolutionary Biology,* in 2001.[8]

This study into "the level of population differentiation among different populations of human genes"[9] with regard to different biological processes found that the "genes involved in osteoblast [cells with a single nucleus that synthesize bone] development were identified as being enriched with higher FST SNPs, a result consistent with the proposed role of the skeletal system in accounting for variation among human populations."[10]

In other words, the scientists detected that different genetic structures at SNP level produced different skeletal structures.

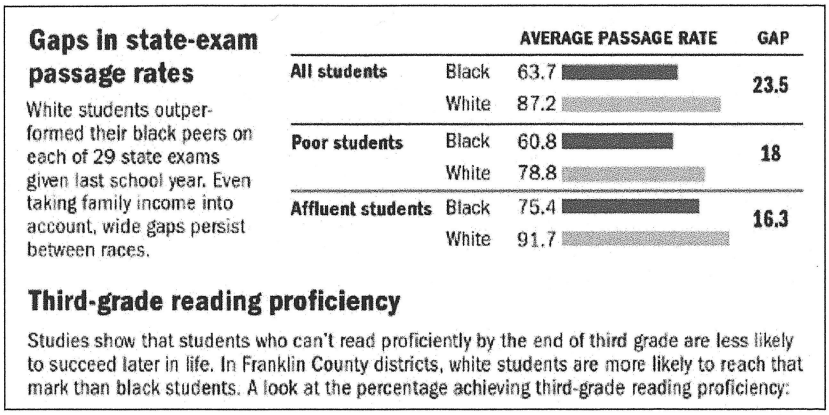
In addition, the study found that "genes involved in the development of hair follicles, where hair is produced, were also found to have higher levels of population differentiation, consistent with hair morphology being a distinctive trait among human populations."[11]

"Other genes that showed higher levels of population differentiation include those involved in pigmentation, spermatid, nervous system and organ development, and some metabolic pathways, but few involved with the immune system."[12] Race is therefore a biological state which is clearly far deeper than "just skin color."

5. "Racial differences in intelligence are explained by socioeconomic factors."

The reality is that multiple studies have repeatedly show that even when income, parenting, and all other social factors are accounted for, the achievement gap between Africans and all other races persists.

8 "Different Level of Population Development among Human Genes," Wu, D.D., et al., *BMC Evolutionary Biology*, 11 (2011):16.
9 *Ibid.*
10 *Ibid.*
11 *Ibid.*
12 *Ibid.*



Gaps in state-exam passage rates

White students outperformed their black peers on each of 29 state exams given last school year. Even taking family income into account, wide gaps persist between races.

		AVERAGE PASSAGE RATE	GAP
All students	Black	63.7	23.5
	White	87.2	
Poor students	Black	60.8	18
	White	78.8	
Affluent students	Black	75.4	16.3
	White	91.7	

Third-grade reading proficiency

Studies show that students who can't read proficiently by the end of third grade are less likely to succeed later in life. In Franklin County districts, white students are more likely to reach that mark than black students. A look at the percentage achieving third-grade reading proficiency:

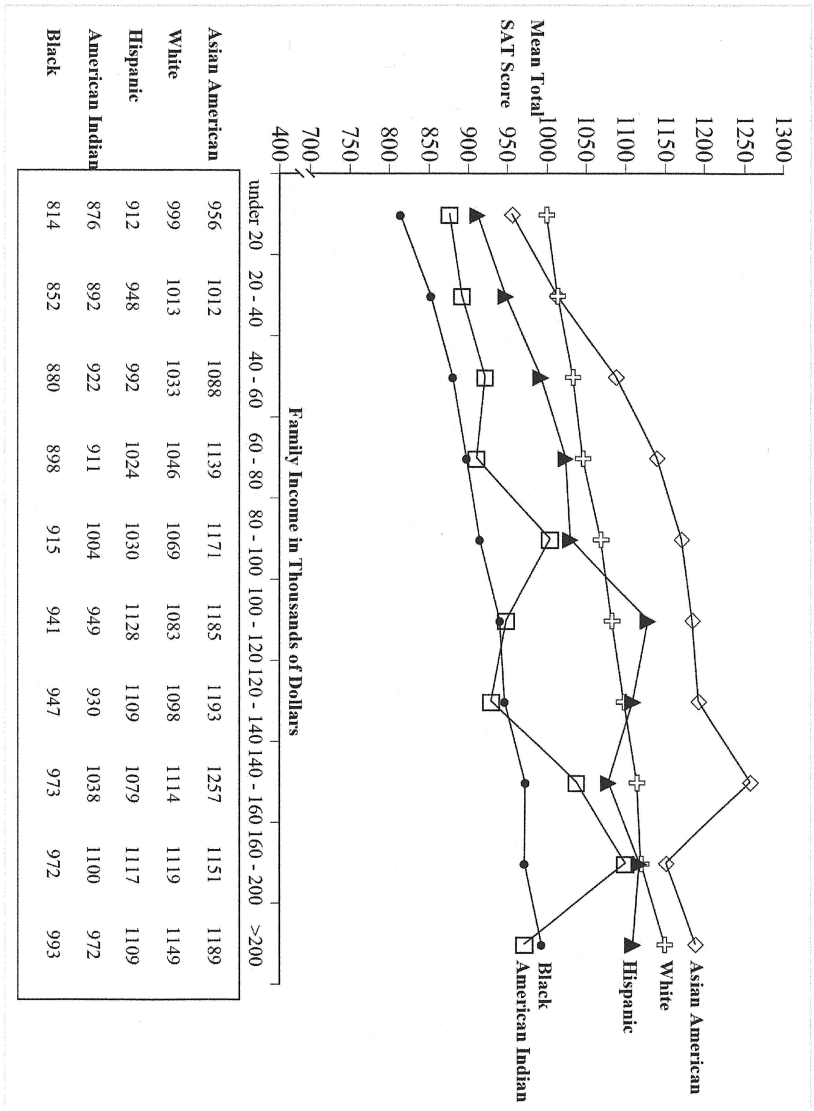
"*Wide racial gap persists in education testing*," The Columbus Dispatch, *September 22, 2013.*

Right: "Family Income Differences Explain Only a Small Part of the SAT Racial Scoring Gap," The Journal of Blacks in Higher Education, *January 22, 2009.*

At All Family Income Levels, There is a Persisting Racial Gap in SAT Scores

Family Income	Mean Black Score	Mean White Score	Racial Gap
Under $20,000	798	978	180
$20,000 to $40,000	836	995	159
$40,000 to $60,000	864	1017	153
$60,000 to $80,000	889	1032	143
$80,000 to $100,000	908	1052	144
$100,000 to $120,000	922	1066	144
$120,000 to $140,000	926	1073	147
$140,000 to $160,000	942	1087	145
$160,000 to $200,000	943	1091	148
More than $200,000	981	1130	149

Source: The College Board



	under 20	20 - 40	40 - 60	60 - 80	80 - 100	100 - 120	120 - 140	140 - 160	160 - 200	>200
Asian American	956	1012	1088	1139	1171	1185	1193	1257	1151	1189
White	999	1013	1033	1046	1069	1083	1098	1114	1119	1149
Hispanic	912	948	992	1024	1030	1128	1109	1079	1117	1109
American Indian	876	892	922	911	1004	949	930	1038	1100	972
Black	814	852	880	898	915	941	947	973	972	993

Table 12: Mean Total SAT Scores (Critical Reading + Mathematics) for North Carolina by Family Income and Racial/ Ethnic Group, 2009–2010. Public schools of North Carolina, "The North Carolina 2010 SAT Report."

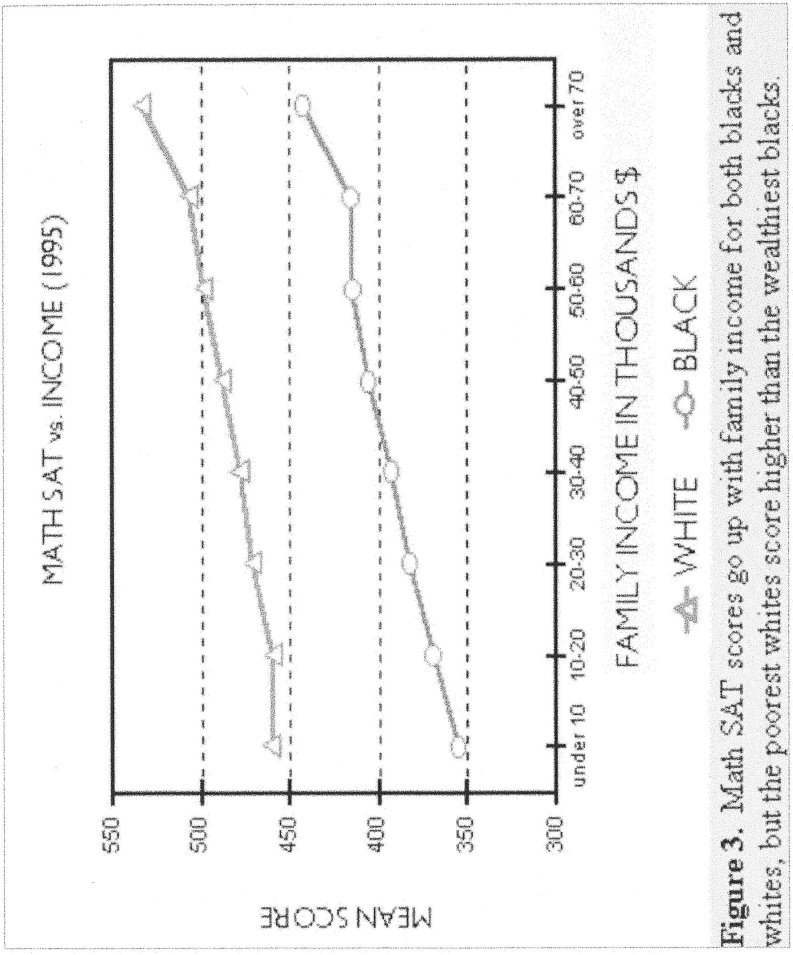


Figure 3. Math SAT scores go up with family income for both blacks and whites, but the poorest whites score higher than the wealthiest blacks.

"Standardized Tests: The Interpretation of Racial and Ethnic Gaps," La Griffe du Lion, *Volume 2, Number 3, March 2000.*

Table 2. Mean SAT Mathematics and Verbal Scores by Family Income for the 2003 College Bound Cohort

	Black Test-takers		White Test-takers		Total Sample	
Family Income	Math Score	Verbal Score	Math Score	Verbal Score	Math Score	Verbal Score
Less than $10,000	382	381	478	480	418	418
$10,000 to $15,000	395	398	478	481	435	439
$15,000 to $20,000	400	405	485	488	446	450
$20,000 to $25,000	409	413	493	495	460	463
$25,000 to $30,000	411	419	495	497	466	470
$30,000 to $35,000	419	426	502	504	479	482
$35,000 to $40,000	422	430	504	505	484	487
$40,000 to $50,000	431	438	510	510	496	498
$50,000 to $60,000	441	450	516	514	507	506
$60,000 to $70,000	440	450	521	519	512	512
$70,000 to $80,000	448	457	528	524	521	518
$80,000 to $100,000	461	468	539	534	533	529
More than $100,000	490	495	568	557	564	555
No Response						

Table 2. Mean SAT Mathematics and Verbal Scores by Family Income for the 2003 College Bound Cohort: from "Race, Poverty and SAT Scores: Modeling the Influence of Family Income on Black and White High School Students' SAP Performance," Dixon-Roman, Ezekiel et al., Teachers College Record, Volume 115 Number 4, 2013, 1–33.

Afterword

There can be little doubt that, left free and independent, scientists will over the course of time uncover even more secrets locked in the human genome, and subsequently produce even more genetic evidence of racial diversity.

From the preceding chapters, it is evident that the science of DNA has provided definitive proof that race is firmly rooted in biology, and that racial attributes are inherited traits.

The denial of this reality is one of the most important sociological phenomena of modern times. Rooted in an intellectually barren and factually devoid wilderness, race-denial exists due to endless repetition in the establishment media, the defamation of those who dare speak out, and the censoring of all facts.

Any society which fails to take biology into account in its social ordering will pay the price. Nothing is greater than the laws of nature and biological fact, and any attempt to flout those realties will end in disaster.

May all people with goodwill and understanding accept this reality, and act accordingly.

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